Internists' Supplement

Contributed by

The American College of Osteopathic Internists

These papers, unless otherwise indicated, were presented at the annual meeting in Philadelphia, October 7-10, 1954.

Volume 10
Number 1

February, 1955
Pages 1 to 35

INTERNISTS' SUPPLEMENT

American College of Osteopathic Internists

Editorial Office, 212 East Ohio St., Chicago 11.
Address all communications to the Chicago office.
Copyright, 1955, by the American Osteopathic Assn.

EDITORIAL COMMITTEE

LEONARD V. STRONG, JR., CHAIRMAN.....New York
MAXWELL R. BROTHERS.....Los Angeles
RICHARD DE NISE.....Des Moines, Iowa

OFFICERS

PRESIDENT.....Neil R. Kitchen, Detroit
VICE PRESIDENT.....Earl F. Riceman, Philadelphia
SECRETARY-TREASURER.....Glennard E. Lahrson, Oakland, Calif.

Volume 10 **February, 1955** **Number 1**

CONTENTS

Intermittent Positive Pressure Breathing in Chronic Diseases of the Lung: The A. D. Becker Memorial Lecture, 1954.
Orville L. Hastings, D.O., Long Beach, Calif. 3

Atherosclerosis: The Ralph L. Fischer Memorial Lecture, 1954.
Charles M. Worrell, D.O., Harrisburg, Pa. 6

The Treatment of Acute Nephritis.
Richard P. De Nise, D.O., Des Moines, Iowa 11

Clinical Observations on the Herniated Intervertebral Lumbar Disk.
James M. Eaton, D.O., F.A.C.O.S., Philadelphia 13

Diagnosis and Treatment of Cancer of the Colon.
Arthur M. Flack, Jr., D.O., F.A.C.O.S., Philadelphia 17

Anesthetic Evaluation and Management of the Poor-Risk Patient.
Francis J. Smith, D.O., F.A.C.O.A., Philadelphia 21

Nutritional and Electrolyte Preparation of the Poor-Risk Surgical Patient.
Morton Terry, B.A., D.O., M.Sc., Miami 25

Diagnosis of Pancreatitis.
M. T. Gutensohn, D.O., Kirksville, Mo. 29

The Diagnosis of Infectious Hepatitis.
Robert Sedar, D.O., Denver 31

The Nephrotic Syndrome in Children.
Leo C. Wagner, D.O., Lansdowne, Pa. 33

Intermittent Positive Pressure Breathing in Chronic Diseases of the Lung

The A. D. Becker Memorial Lecture, 1954*

ORVILLE L. HASTINGS, D.O.
Long Beach, Calif.

First of all, may I express my profound appreciation to you for the honor which you have bestowed upon me in inviting me to deliver the Becker Memorial Lecture. To be asked to address you on this occasion, when we are honoring the memory of such an outstanding physician as Dr. Arthur D. Becker, a beloved former president of the A.C.O.I., gives me a special feeling of gratification.

I have elected to address you today on a topic of particular interest to me, the use of intermittent positive pressure breathing in the treatment of chronic lung diseases. I have used this method for 3 years in treating 300 patients with chronic pulmonary disease. It is estimated that these patients, derived from private practice and the Los Angeles County Osteopathic Hospital, have received 2,500 treatments.

Much of the disability in many chronic pulmonary diseases is the result of disturbed pulmonary physiology rather than of organic diseases.[1-3] This pulmonary physiologic disturbance centers about obstruction of the airway with subsequent impairment of the breathing mechanism and bronchial drainage and the eventual development of hypoxia.[2,4,6,7] Airway obstruction, whether the mechanism is a partial obstruction, a check-valve, or total obstruction, results in accumulated secretions with superimposed infection, mucosal edema, mural thickening, and varying degrees of bronchospasm.[2,8]

One of the successful therapeutic measures for the relief of bronchospasm, pulmonary infection, impaired breathing mechanics, and hypoxia is to combine intermittent positive pressure breathing simultaneously with nebulization of a bronchodilator drug.[1,2,9-12]

There was no selection of patients other than not including those who might be harmed by the procedure. The contraindications for intermittent positive pressure breathing during inspiration were shock and recent hemoptysis.

Before treatment a careful study was made of all patients, including a complete history, physical examination, hemograms, sedimentation rate, urinalysis, serologic tests for syphilis, electrocardiogram, chest fluoroscopy, and chest roentgenograms. Many of the chest roentgenograms were made during expiration as well as inspiration. Such studies as circulation time, bronchoscopy, tomography, bronchography, and bronchial and sputum cultures were performed when indicated. Vital capacity determinations were made before therapy in all patients and in 50 per cent after their treatment. Maximal breathing capacity determinations and spirograms were not made, but the research findings of Motley and Tomashefski[1] and Cournand and his associates[5] were accepted.

Therapeutic effects were evaluated with repeated physical examinations, laboratory studies, roentgenograms, vital capacity determinations, fluoroscopic studies of the chest before, during, and after therapy, by studying the effect of the treatment on symptoms, and from the effect on the pulmonary arterial pressure as determined by intracardiac catheterization before and after the procedure. The Bennett pressure therapy unit (model TV-2P) was used to treat all the patients. A gas and any desired aerosol are simultaneously administered. The pressure characteristics of this unit provide a slow rising of the inspiratory pressure and a rapid falling of the expiratory pressure which promote the movement of secretions from the smaller bronchi of the larger bronchi and trachea. The instantaneous flow rate is sufficiently high for all patients with dyspnea. Others have reported on the physiologic effects of this and similar equipment.[5, 12, 13] Intermittent positive pressure used in this way does produce a small decrease in cardiac output;[13, 14] however, this reduction is less than that which normally occurs in man when changing from the supine to the standing position.[12, 15]

It has been my clinical observation that the effect of pressure breathing on cardiac output seems to be in direct ratio to the mean pressure. The greater the mean pressure, the greater is the effect on cardiac output. The mean pressure can be lowered mechanically by intermittently permitting the intrapulmonary pressure to drop to an ambient pressure. To accomplish this the duration of expiration should be longer than the period of inspiration to allow the heart to "catch-up" during this period of phasic decreases in intrapulmonary pressure.

If the cardiac output were noticeably decreased as the result of intermittent positive pressure during inspiration, the pressure within the right ventricle should be increased as the result of the increased venous pressure. However, intracardiac catheterization failed to confirm this finding. The pressure reading within the right ventricle before intermittent positive pressure breathing during inspiration was 44/8 mm. of mercury; the pressure reading within the right ventricle after treatment was reduced to 30/5 mm. of mercury.[16]

Aerosols of various medications and 100 per cent oxygen were used in all treatments. Bronchodilators, antibiotics, and wetting agents were the types of aerosols used. Equal success was obtained with Isuprel hydrochloride (1:200 solution) and racemic epinephrine solution (Vaponefrin), both potent bronchodilators. Elevation of the blood pressure or pulse rate was not appreciably altered in any patient. Twelve to 15 drops of the bronchodilator were administered in each treatment. At the beginning of the survey, only 4 to 6 drops of the bronchodilator were used, but it was felt that the increase offered additional relief from associated bronchospasm.

Penicillin and, occasionally, streptomycin were included in the aerosol treatment. The dosage of the antibiotic varied with the severity of the respiratory tract infection and bacterial sensitivity. The average dose of penicillin was 50,000 units, and the average dose of streptomycin was 0.5 gram.

Cetyl pyridinium (Ceepryn) chloride was the wetting agent which was used. Ceepryn reduces surface tension and promotes loosening of bronchial secretions. It also has a mild bacteriostatic effect. For those pa-

tents who had difficulty in raising sputum, 4 drops of a 1:1,000 solution of Ceepryn were added to the aerosol mixture.

The specific method of treatment follows: The agents to be aerosolized and 1 cc. of isotonic sodium chloride solution were placed in the nebulizer. A mask or rubber mouthpiece was used to attach the machine to the patient's mouth; most patients found the mask more comfortable. Following application of the mask or mouthpiece, the line of control pressure was adjusted. A pressure of 10 to 20 cm. of water was tolerated by all the patients except those in status asthmaticus. In these patients, it was preferable to approach the average pressure very gradually. Higher pressures were usually necessary in those patients with pulmonary fibrosis. The nebulizer was adjusted to produce a true aerosol. The average duration of treatment was 20 minutes. Children were prone to fatigue at 10 to 15 minutes.

Each patient was encouraged to breathe slowly and deeply. The instantaneous flow rate was high enough for patients who evidenced hyperventilation and who had a large tidal volume. The flow-sensitive valve permitted patients with emphysema to exhale at will before a new inspiratory cycle began. This respirator, unlike most automatic respirators, follows the slightest breathing effort of the conscious subject since the cycling pattern is not fixed. These features permitted easy, comfortable, and almost effortless breathing in the conscious patient if a suitable type of face mask or mouthpiece was used. Neither the patient nor the breathing unit was harmed by unavoidable coughing. The frequency of treatments varied according to the patient. The more frequently the patient could have treatment, the sooner he reached a plateau, at which time the number of treatments was gradually reduced. As long as improvement was maintained, the frequency of treatment continued to be reduced until therapy could be discontinued entirely. The patients with bronchiectasis or chronic pulmonary emphysema were advised to return every month for 2 to 4 days of treatment.

The results of treatment were evaluated clinically. The clinical evaluation included the patient's subjective feelings as to changes in cough, expectoration, and exercise tolerance, and a chest examination for rales and wheezes. The subjective relief consisted primarily of less tightness of the chest, lighter and easier breathing, decreased secretions and cough, improved appetite, and increased strength. Usually for the first 2 or 3 days after the beginning of treatment the amount of secretions was increased. After approximately a week of treatment the secretions dwindled and, in some cases, totally disappeared. Undoubtedly the most beneficial effect of intermittent positive pressure breathing with a vasodilating agent was the promotion of drainage of the bronchi and bronchioles.

Objective clinical evaluation included vital capacity determinations, fluoroscopic study of diaphragmatic motion, and right ventricular pressure determinations obtained by cardiac catheterization. In many instances the timed vital capacity determination method was used; its technic is described elsewhere.[17] In 90 per cent of the cases, the vital capacity was increased. The individual increment in vital capacity was as low as 100 cc. and as high as 1,500 cc. Blood pressure was temporarily reduced 10 to 30 cm. in all cases.

From the experience gained with this respirator on the over-all problem of disturbed pulmonary physiology, it appears possible to predict the effect of intermittent positive pressure breathing during inspiration on certain specific medical conditions.

In the early and moderate stages of emphysema, intermittent positive breathing during inspiration was notably effective. In this stage of emphysema[5, 18] bronchospasm and edema of the terminal bronchioles predominate. One hundred emphysematous patients were treated, fifty-one of whom were classified as having an early type of chronic pulmonary emphysema. All of these patients showed improvement in color owing to better oxygenation. The symptoms of cough decreased noticeably in most cases, and expectoration was less difficult. The breathing pattern became more regular and exercise tolerance was increased in 85 per cent of the cases. Diaphragmatic mobility increased in all patients.

In the advanced stage of emphysema, when there is a marked loss of alveolar elasticity with uneven ventilation, rupture of the walls, and obliteration of the capillaries, the use of intermittent positive pressure breathing during inspiration was not as effective. However, patients reported that they felt better and that their breathing pattern was more regular, but their exercise tolerance remained stationary. Fluoroscopy failed to reveal increased ventilation in the lungs in this group of advanced emphysematous patients. Six of eleven patients in this group purchased their own respirator for home use. The follow-up study on these patients revealed that as long as the patient had at least one daily treatment he was not incapacitated.

In thirty-eight patients with moderate to advanced emphysema, bronchial lavage with normal saline was performed initially, with the aid of bronchoscopy, to relieve the retention of viscid secretions. The bronchoscopic procedure was then followed by intermittent positive pressure breathing with only palliative results. In three of these patients, the use of exsufflation with negative pressure created a more prolonged and adequate elimination of the mucopurulent secretion than did positive pressure breathing during inspiration. However, the effort expended during this form of treatment was too fatiguing and necessitated a return to the Bennett valve machine. The use of exsufflation with negative pressure is still in a beginning stage, but the studies of Segal and Salomon,[19] Barach and his associates,[20, 21] and Beck and Barach[22] are quite encouraging.

Twenty patients with bronchiectasis, one of whom required a pneumonectomy because of an advanced stage of unilateral, irreversible bronchiectasis, were treated with intermittent positive pressure breathing during inspiration. The results were very good in 90 per cent of this group. Expectoration was easier and there was the usual initial increase in the amount of sputum. The amount of sputum decreased as the treatments were continued, and the cough lessened noticeably or completely. The incidence of pulmonary infections was favorably influenced by this form of treatment. The patients showed increased exercise tolerance. The gratifying response in these bronchiectatic people was due chiefly to promoting better bronchial drainage and expectoration of sputum. Many patients indicated that this therapy was far superior to postural drainage in facilitating the raising of sputum, and that intermittent positive pressure breathing gave them the first real relief they had experienced in many years. Three patients with advanced bronchiectasis required bronchial lavage at weekly intervals for an aver-

age period of 6 months before the use of intermittent positive pressure breathing was noticeably efficacious.

One comatose patient was treated while in a state of respiratory depression and peripheral circulatory failure as a result of barbiturate intoxication. Death ensued during the procedure. Alternative positive-negative pressure (exsufflation with negative pressure) would have been preferable to intermittent positive pressure during inspiration and would have been the procedure had the unit been available.

In bronchial asthma, in which bronchospasm is the major factor contributing to the patient's discomfort, it was anticipated that intermittent positive pressure breathing during inspiration would be beneficial. The clinical results corroborated this assumption. After treatment the patient invariably felt better. Breathing was easier, and there was less wheezing as a rule. Cough was more effective in raising mucus.

One of the most gratifying results with intermittent positive pressure breathing during inspiration was observed in a patient who had alveolar edema as the result of a skull fracture. This patient required five treatments per day for 3 days before he was able to ventilate with ease. Apparently alveolar aeration became more uniform as the result of the decreased residual volume, and subsequent improvement of the ventilation-perfusion relation followed.

SUMMARY

Physiologically directed therapy such as intermittent positive pressure breathing during inspiration involves a procedure which tends to restore or maintain the function of the respiratory system. Chronic diseases of the lungs and bronchi in many cases appear to be largely reversible. Even in cases of hypertrophic pulmonary emphysema, in which a chronic, organic form of dyspnea seems to be present, favorable alteration in the pathologic physiology of the disease may lead to marked improvement in respiratory function.

4320 Atlantic Ave.

REFERENCES

1. Motley, H. L., and Tomashefski, J. F.: Treatment of chronic pulmonary disease with intermittent positive pressure breathing. 1. Evaluation by objective physiological movements. Arch. Indust. Hyg. 5:1-9, Jan. 1952.

2. Smart, R. H., Davenport, C. K., and Pearson, G. W.: Intermittent positive pressure breathing in emphysema of chronic lung diseases. J. Am. M. A. 150:1385-1390, Dec. 6, 1952.

3. Gordon, B., *et al.*: Considerations of clinical and physiologic factors in treatment of chronic pulmonary conditions. Dis. Chest 19: 271-287, March 1951.

4. Westcott, R. N.: Physiology of ventilation in chronic lung disease. Postgrad. Med. 12:329-338, Oct. 1952.

5. West, J. R., *et al.*: Physiopathologic aspects of chronic pulmonary emphysema. Am. J. Med. 10:481-496, April 1951.

6. Cournand, A.: Some aspects of pulmonary circulation in normal man and in chronic cardiopulmonary disease. Circulation 2:641-657, Nov. 1950.

7. Jackson, C., and Jackson, C. L.: Bronchoesophagology. W. B. Saunders Co., Philadelphia, 1950.

8. Hastings, O. L.: Evaluation of tumors within chest. Clin. Osteop. 49:535-543, Aug. 1953.

9. Saklad, M.: Pressure breathing in surgery. J. Thoracic Surg. 28:31-38, July 1954.

10. Segal, M. S., *et al.*: Intermittent positive pressure breathing; its use in inspiratory phase of respiration. New England J. Med. 250:225-232, Feb. 11, 1954.

11. Motley, H. L., Lang, L. P., and Gordon, B.: Use of intermittent positive pressure breathing combined with nebulization in pulmonary disease. Am. J. Med. 5:853-856, Dec. 1948.

12. Motley, H. L., *et al.*: Intermittent positive pressure breathing; means of administering artificial respiration in man. J. Am. M. A. 137: 370-382, May 22, 1948.

13. Cournand, A., *et al.*: Physiological studies of effects of intermittent positive pressure breathing on cardiac output in man. Am. J. Physiol. 152:162-174, Jan. 1948.

14. Motley, H. L., *et al.*: Physiological studies on man with pneumatic balance resuscitator, "Burns model." J. Aviation Med. 17:431-461, Oct. 1946.

15. Hamilton, W. F.: Physiology of pulmonary circulation. J. Allergy 22:397-410, Sept. 1951.

16. Long, W. D.: Personal communication.

17. Segal, M. S., Herschfus, J. A., and Dulfano, M. J.: Simple method for determination of vital capacity—time relationships. Dis. Chest 22:123-129, Aug. 1952.

18. Mounsey, J. P. D., *et al.*: Circulatory changes in severe pulmonary emphysema. Brit. Heart J. 14:153-172, April 1952.

19. Segal, M. S., Salomon, A., and Herschfus, J. A.: Alternating positive-negative pressures in mechanical respiration; cyclic valve device employing air pressures. Dis. Chest 25:640-648, June 1954.

20. Barach, A. L., and Beck, G. J.: Exsufflation with negative pressure; physiologic and clinical studies in poliomyelitis, bronchial asthma, pulmonary emphysema, and bronchiectasis. Arch. Int. Med. 93:825-841, June 1954.

21. Barach, A. L., Beck, G. J., and Smith, W.: Principles and technic of operation of exsufflator. Am. Pract. & Digest Treat. 3:733-738, Sept. 1952.

22. Beck, G. J., and Barach, A. L.: Value of mechanical aids in management of patient with poliomyelitis. Ann. Int. Med. 40:1081-1094, June 1954.

TRENDS IN SUPPORT AND EXPENDITURES FOR MEDICAL RESEARCH

In round numbers, our Nation paid $173 million for medical research in 1952 (the latest year for which analyses are available)—four times the amount spent 12 years ago. The largest share, $73 million or about 42 percent, was the Federal Government's contribution. Medical research, however, came to less than 5 percent of the total investment in all scientific research and development. And Government's 42-percent support of medical research compares with Federal financing of 60 percent of all research.

In this country, substantial public and private backing of medical research dates back to the beginning of this century. Previously, support of learning went mostly to general education, religious teachings, and the humanities. Medical research, however, was not without its champions and donors. As far back as 1801, Dr. David Ramsey, in an address before the South Carolina Medical Society, noted with considerable interest the increase in giving and the beginning of organized research and support in England:

"Two novel institutions of this kind, the first the world has ever seen, reflect equal honor on England and the 18th century. One has been lately instituted for ascertaining, by experiments, the precise effects of the newly discovered elastic fluids. The other for the relief of cancerous patients, in which it is intended to give the utmost scope to medical ingenuity for discovering the best plan of treating that hitherto incurable disease. For this last purpose, one gentleman has lately given £3,000."—Irving Ladimer, *Public Health Reports,* February, 1954.

Journal A.O.A.
Vol. 54, No. 6

Atherosclerosis

The Ralph L. Fischer Memorial Lecture, 1954*

CHARLES M. WORRELL, D.O.
Harrisburg, Pa.

It was not by chance that atherosclerosis was selected as the topic to honor the memory of Dr. Ralph L. Fischer. It was he who stimulated my interest in this subject many years ago in the hope that it could be demonstrated that osteopathic manipulative therapy could arrest the progress of the disease.

REVIEW OF LITERATURE

Atherosclerosis is not an aging disease although it is a disease that is most commonly found in the aged. It may be found in infancy, and many of the aged may be relatively free of the lesions. It varies with race,[1] family,[2] age,[3] sex,[4] nutrition,[5] and profession.[6]

Moon and Rinehart[3] studied the coronary arteries of 250 individuals dying suddenly from traumatic or natural causes. They found that many different histopathologic processes participated in the production of atherosclerosis:

1. Subendothelial fibroblastic proliferation of the intima
2. Increased amounts of mucoid ground substance in the intima and media
3. Fragmentation and degeneration of elastic tissue
4. Formation of collagen fibers in the intimal plaques
5. Regeneration of elastic tissue
6. Deposition of lipid and cholesterol
7. Hyaline degeneration of fibrous connective tissue
8. Deposition of calcium
9. Intramural hemorrhage and fibrosis.

In the early stages, occurring generally in the first or second decade of life, lipids were usually absent but mucoid ground substance was noted in the intima and media in increased amounts. There was proliferation of the subendothelial fibroblasts and small areas of degeneration of the intimal elastic membrane.

In the moderately advanced state, lipids were present in greatest abundance at the base of the plaque and least along the endothelial border, and the plaques were encroaching on the media. The intimal elastic membrane showed greater fragmentation.

In the far advanced state, the bases and centers of the plaques were hyalinized, and here the lipid deposits were greatest, often present in large globules. Stenosis and thrombosis of the lumen were often apparent, and in these arteries, hemorrhage could be demonstrated into the plaque.

These authors believed that the lipid deposits were present not because of imbibition through the endothelial lining of the intima but rather that the deposits followed the route of the vasa vasorum, and they suggested the possibility that an abnormal polymerization of the mucoid ground substance, and not lipid deposition, is the initiating factor. But it is apparent that in order to have the occlusive phenomena of arteriosclerosis, lipid deposition must be present.

Early investigators[7-9] pointed the finger of suspicion at cholesterol as the causative agent, and when Steiner and his associates[10] demonstrated the experimental production of atherosclerosis in the dog by simultaneous administration of cholesterol and thiouracil, much interest was developed and all forms of diet and fat solvents or digestants were investigated, and a diet low in cholesterol became the vogue.

Gertler, Garn, and White,[11] in making a dietary history of healthy males and of survivors of myocardial infarction, found no difference in the cholesterol intake of either group.

Keys[12] found that the low cholesterol diet had no influence on the blood cholesterol level as long as the daily fat intake was elevated, but when both were lowered, the blood cholesterol level was reduced. He also found that a high cholesterol diet did not alter this level if total fats were maintained at a minimum.

Pollack[13] studied diet from another angle. He observed that sitosterol is not absorbed by man; and when it is united with cholesterol, either exogenous or endogenous, it will prevent either its absorption or resorption, whichever the case may be, and will thus reduce the blood level. It is a very distasteful preparation, however, and must be given in excessive amounts and continuously to be effective.

While Gofman and his coworkers[14] have found that a diet low in cholesterol reduces the level of lipoprotein molecules of the S_f 10-20 class, such a diet has not been found to have any consistent relationship to the blood cholesterol levels. Indeed, Aldersberg,[15] in an unpublished report, has stated that it actually increases the synthesis of cholesterol.

Gofman and his group[14] used the ultracentrifuge to investigate these lipoprotein molecules. They found that certain molecules, when in a centrifuge at a force thousands to millions of times the force of gravity, undergo sedimentation in a solution of lesser density or undergo flotation if it is of greater density. They found that this followed a definite pattern, the unit of which they termed the "S-unit" in deference to Svedberg who invented the machine used.

They found that one lipoprotein molecule was most constant in survivors of myocardial infarction and in those susceptible to atherosclerosis and was absent or definitely reduced in animals or humans who because of age or other factors were supposedly immune. This group of lipoproteins had a flotation rate of 10 to 20, the basis for the term S_f 10-20.

They found that these were elevated in diabetes, nephrosis, familial xanthomatosis, coronary insufficiency, hypertension, hypothyroidism, and in survivors of myocardial infarction, and low in children, immune animals, women in the childbearing period, and those on a low cholesterol diet for a long period.

One point of criticism of their figures, however, is the wide variation between the extreme of their cases.

The hormonal influence on the pathogenesis of atherosclerosis has interested investigators for quite some time since it has been repeatedly demonstrated

*Presented at the Eastern Study Conference of the American College of Osteopathic Internists, Philadelphia, March 12, 1954.

that women show a high degree of immunity up to the age of 45.

Wuest, Dry, and Edwards[16] made a careful study of the degree of atherosclerosis in the coronary vessels of forty-nine women who had had bilateral oophorectomy from 2 to 42 years prior to death. They compared these with controls of 600 men and of 680 women of comparable age whose ovaries had not been removed. The average sclerosis was greater in the oophorectomized women than in the female controls but was less than in the male controls. However, when they were grouped according to the grade of sclerosis, the number of women past the age of 65 exceeded the number of men in Grade III or higher, in other words, those highly susceptible to myocardial infarction.

Rivin and Dimitroff[17] studied autopsy records of men with carcinoma of the prostate who had been treated with estrogens, and of castrated women and women with breast carcinoma. They found that males treated with estrogen of sufficient potency to produce gynecomastia had much less atherosclerosis than the controls, and women with breast carcinoma, in whom it was presumed that hyperestrogenism was present, had much less atherosclerosis than the normal female controls. They postulated that not only does estrogen protect the individual but it actually reverses the atherosclerotic process.

Another investigation was that of Barr[18] who used Cohn's microfractionation method number 10 for chemical analysis of lipoproteins. He found that the most important factor from the standpoint of constancy was the relation of total cholesterol in alpha-lipoprotein to the total beta-lipoprotein and that in normal women from 18 to 35 years of age, the ratio was 34 to 61 compared to that of 25 to 72 in normal men, but in women 45 years or older, the ratio paralleled that of the men. The ratio is less favorable in survivors of myocardial infarction and other lipid diseases such as nephrosis, diabetes, and familial xanthomatosis.

While the studies of S_f 20 by Gofman and his associates[14] showed many normal individuals had high 12-20 levels and a fair percentage of survivors of myocardial infarction had normal levels, Barr's studies found the percentage of cholesterol in the form of alpha-lipoprotein of normal women was satisfactorily elevated. This was also demonstrated to a lesser degree in normal men and was rare in survivors of myocardial infarction. He found the same high level of alpha-lipoprotein in animals that are immune to atherosclerosis.

Acting on these findings, Barr[18] then gave Estinyl or Premarin, in doses of 10,000 I.U. daily, to a group of survivors of myocardial infarction. He noted that in every case there was an increase of alpha-lipoprotein and a decrease of beta-lipoprotein.

There was also a fall in the cholesterol-phospholipid ratio and, in many cases, a fall in total cholesterol. When the medication was discontinued, the levels reverted to the original. He also noted that administration of testosterone reversed the effects of estrogen. When testosterone was given in daily doses of 50 mg., the alpha-lipoprotein was diminished far below its level at the start of Estinyl therapy, and even when testosterone was reduced to 10 mg. daily, the original improvement was lost.

Stamler, Pick, and Katz[19] found rather different hormonal effects on the actual atherosclerotic process in experimental studies of cockerels fed a high cholesterol diet. They divided the birds into six groups, the first being the control group, and the others being given, respectively, estrogen, androgen, estrogen and androgen, gonadotropin, and estrogen and gonadotropin.

They found that 75 per cent of the control group developed atherosclerosis in 10 per cent of the coronary arteries. With estrogen, 15 per cent developed atherosclerosis in 2 per cent of the coronary arteries, but when estrogen and androgen were combined, there was no atherosclerosis in any of the arteries.

Barr[18] in his studies has proved that the lipoprotein content of the blood is distinctly affected by sex hormones, and, from the standpoint of atherogenesis, it is adversely affected by testosterone.

Katz,[20] however, has found that in experimental atherosclerosis the use of testosterone with estrogen distinctly inhibits atherogenesis.

CLINICAL STUDIES

Since the large doses of Estinyl used by Barr caused unfortunate side reactions, principally feminizing characteristics, an attempt has been made to study a group of patients clinically, using Estinyl and Depo-testosterone. These patients were known to be atherosclerotic by having had attacks of acute coronary occlusion or by having the anginal syndrome in which there are electrocardiographic changes that demonstrate coronary insufficiency.

Some authorities believe that the ballistocardiogram will detect the changes of coronary artery disease earlier than other laboratory modalities. For this reason, this instrument was employed in this research study.

The patients were instructed to take 0.25 mg. of Estinyl daily and were given 50 mg. of Depo-testosterone every 2 weeks. Ballistocardiograms were taken at monthly intervals. The patient was instructed to eat at least 2 hours prior to the study and not to smoke for a period of ½ hour before this time. He was then placed on an x-ray table to establish a firm base. A small kapok pillow was permitted for the head. A minimum of 20 minutes of complete rest on the table was employed to establish a basal level.

The equipment used was a Dock electromagnetic ballistocardiograph attached to a Cambridge string galvanometer electrocardiograph. In the early studies, the timing was recorded with the stethograph, but later this was augmented by a pulsometer.

A total of 16 patients were started on the project, 10 of whom had had acute coronary occlusion. None of these was utilized until he had been ambulant for a month because of the usual transition attending this state.

Three patients had the anginal syndrome with electrocardiographic changes of coronary insufficiency. Three were hypertensive with diastolic pressures above 120 mm. of mercury, x-ray evidence of increase in heart size, and/or electrocardiographic changes that were characteristic of left heart strain. Five discontinued the project for the following reasons: two because of indifference—they did not report after the first time; one because of the return of menstruation; one because of a return of peptic ulcer symptoms; and one because of the development of a severe cardiac neurosis.

The criteria employed to classify coronary artery disease according to the ballistocardiograms were those projected by Brown and his associates:[21]

Grade 1. The regularity and definitiveness are preserved. The inspiratory I-J amplitude is normal for the minute volume. The expiratory complexes vary, however, in that there is an abnormally decreased amplitude of the waves, and the complexes usually vary in definitiveness. This accounts for the increased [abnormal] R.V.I.

Grade 2. One half or more of the complexes are abnormal, again mainly during expiration. The minute volume in this group is usually decreased owing to a corresponding decrease in the amplitude of the inspiratory complexes as well.

Grade 3. The complexes in both inspiration and expiration show varying degrees of abnormality in regularity and definitiveness. The complexes are still individually identifiable. The amplitude of all the complexes is low.

Grade 4. Totally abnormal complexes are present throughout in this group. They are of low amplitude and are unidentifiable.

The results of the study were indecisive but none the less encouraging. For the study to be of real value, this group should be studied for a 6-month period without therapy and for another 6 months with estrogen alone.

The ballistocardiograms (Figs. 1-11) show the findings at the beginning of treatment and after varying periods. The shortest interval was 2 months and 3 days and the longest 5 months.

Four of the eleven cases showed little or no improvement in their complexes. Five improved from grade 4 to grade 3. Of these, four were males. The female, during the course of the project, developed acute cholecystitis that necessitated surgery. This was completed and recovery was uneventful, but for 2 weeks following operation, treatment was discontinued. It is to be noted that at the time of withdrawal, menstruation occurred for the first time in 11 years. Four

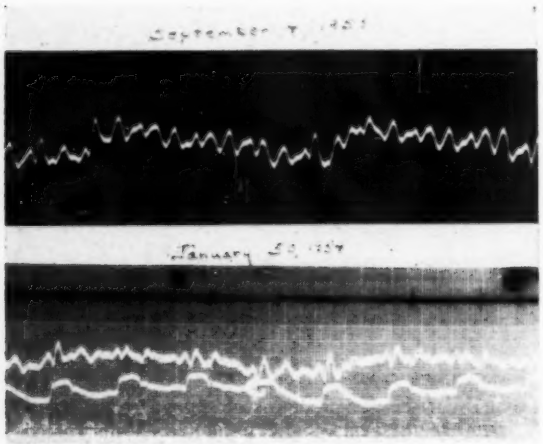


Fig. 1. Ballistocardiogram taken on September 4, 1953, was poor Grade 3, with H-I-J complex slurred and L wave as prominent as J wave. January 30, 1954, ballistocardiogram was Grade 1, with H-I-J complex distinct. L wave was still quite prominent, but all complexes were recognizable.

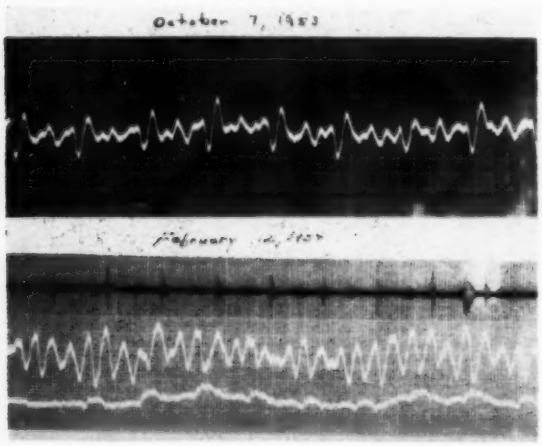


Fig. 2. Ballistocardiogram taken on October 7, 1953, shows fused H-I-J complex and extremely prominent L with very deep K; complexes were recognizable. On February 12, 1954, ballistocardiogram none of the complexes were recognizable. H-I-J complex was distinctly fused, and L wave was more prominent than J.

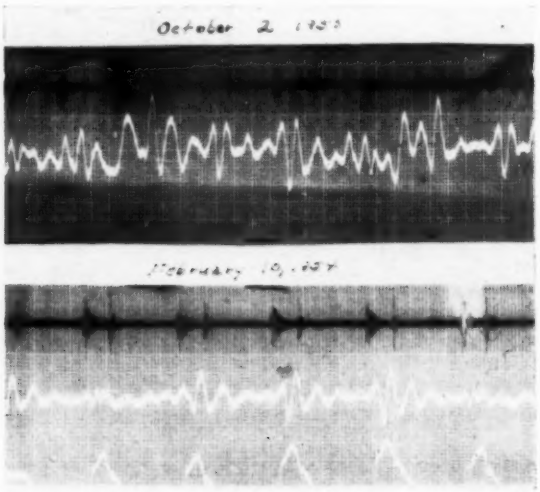


Fig. 3. On October 2, 1953, few of the complexes were identifiable with H-J fused and L more prominent than J; February 10, 1954, all complexes were identifiable, but L was markedly prominent and H-J was fused (low Grade 3 to early Grade 1.) This patient had a coronary occlusion with congestive heart failure and bigeminal rhythm. In the past year he has developed Paget's disease of the bone, and it was for this reason he was placed on estrogen therapy.

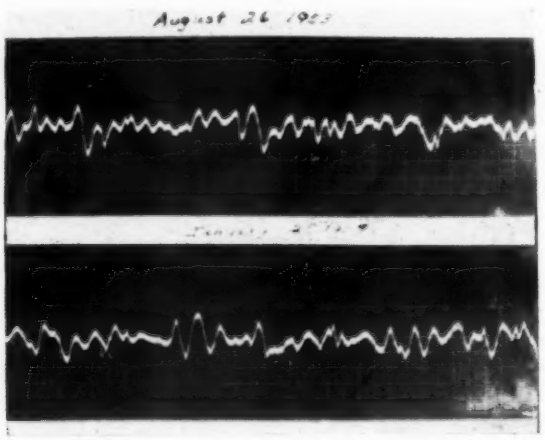


Fig. 4. On August 26, 1953, most complexes were unrecognizable, with L a prominent portion of complexes; January 25, 1954, H-I-J was fused with L still being the most prominent (Grade 4 with no improvement).

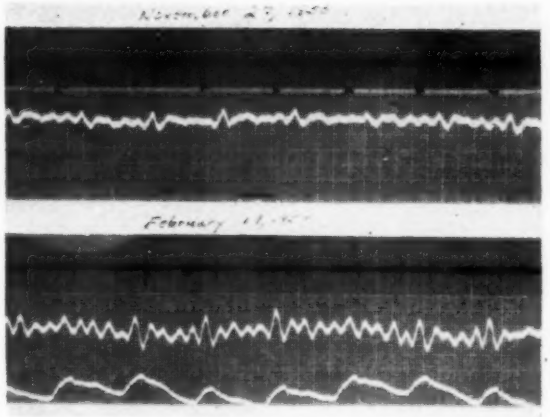


Fig. 5. November 27, 1953, the complexes were very small with H-J fused and L wave the prominent feature; February 19, 1954, about half the complexes were recognizable and L was quite prominent. Amplitude was distinctly increased (Grade 4 to Grade 2).

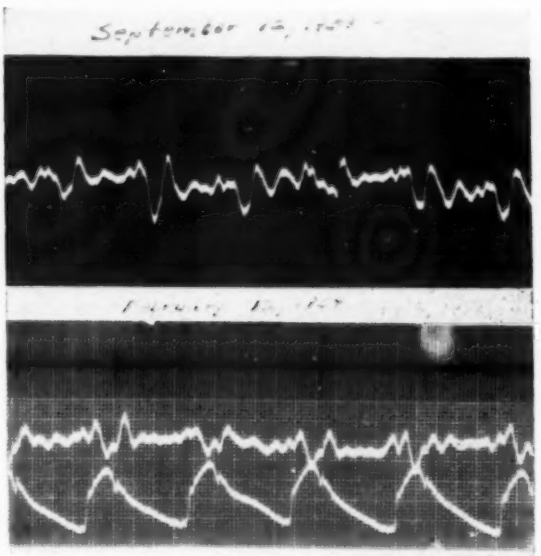


Fig. 6. On September 12, 1953, H-I-J complex was fused in many complexes, K wave prominent, and L more prominent than J; on February 12, 1954, H-I-J was identifiable in most complexes, but somewhat slurred in the third. L was still prominent but complexes identifiable (Grade 3 to Grade 2).

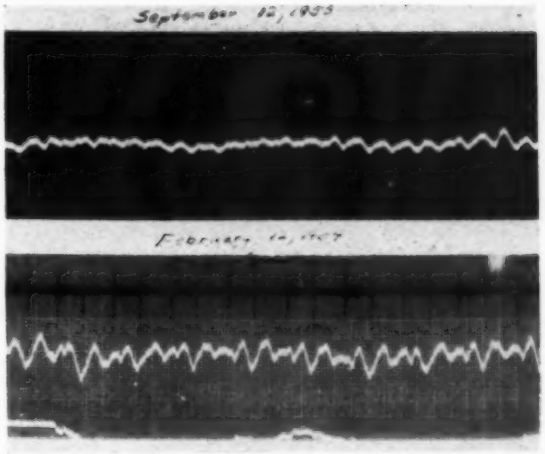


Fig. 7. On September 12, 1953, the complexes were very low and none were identifiable; on February 12, 1954, H-I-J was identifiable in most complexes, but fused in some; L was still the most prominent feature but K had depth (poor Grade 4 to Grade 3).

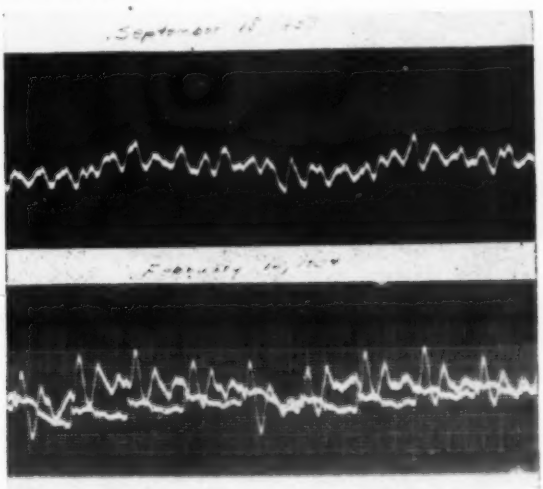


Fig. 8. On September 18, 1953, H-I-J was fused, with K wave the most prominent and slurred. On February 12, 1954, J-K was very prominent with K more prominent than L; all complexes were identifiable (Grade 4 to Grade 1).

patients were survivors of myocardial infarction, and one was hypertensive. It is noteworthy that the fixed blood pressure of this patient dropped from an average of 220/140 to 170/110. This drop was not noted, however, in a female hypertensive who was studied.

Two showed quite dramatic improvement. One, the hypertensive female mentioned above, improved remarkably clinically, and her failure to restrict her activities may in part account for her unchanged blood pressure. The other, a male with electrocardiographic changes and anginal syndrome, was just as remarkably improved clinically.

The side-effects were breast engorgement in all males; at times, itching of the nipples; and, in a few cases, breast tenderness.

One patient complained of actual pain. He was also completely devoid of libido and showed absolutely no improvement. The fact that he had had two known infarctions, both of which occurred while his wife was institutionalized and that she is now home, may be highly contributory. One other patient complained of reduction of libido. Both of these men would be classed as effeminate.

Several patients complained of nausea, but this was corrected by giving Estinyl on retiring. Two had recurrence of menses, one after 1 year (she discontinued treatment because of this) and one after 11 years. Another patient had recurrence of ulcer symptoms and discontinued early in the project.

SUMMARY

1. The atherosclerotic process may occur very early in life and is not initiated by lipid deposits. Luminal stenosis and occlusion, however, are dependent upon the deposition of cholesterol.

2. This deposition is not caused by elevated cholesterol blood levels but it may be caused by other lipoprotein constituents such as molecules of the S_f 12-20

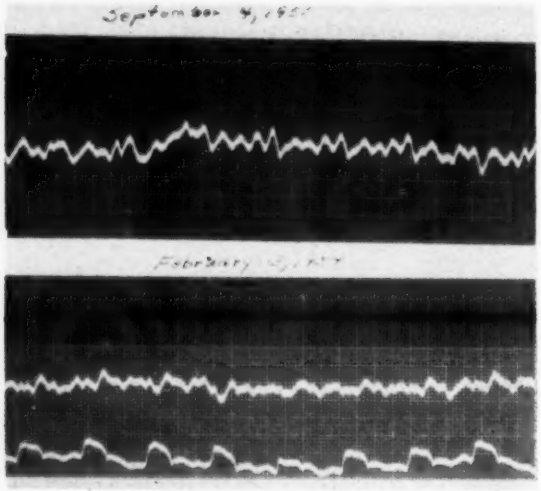


Fig. 9. On September 4, 1953, complexes were unrecognizable with fused H-J and L more prominent than J. February 3, 1954, ballistocardiogram showed no improvement with H-I-J still somewhat fused and L most prominent (Grade 4).

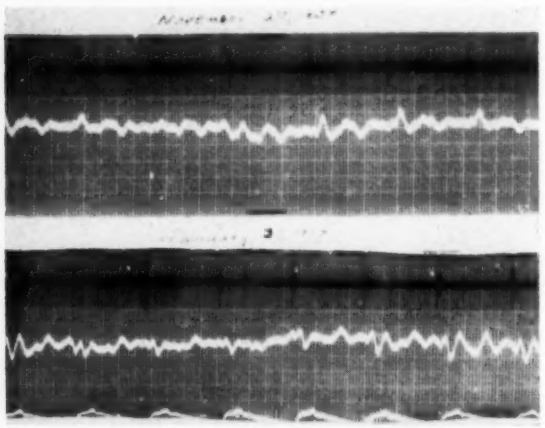


Fig. 10. On November 27, 1953, ballistocardiogram showed fusion of H-I going into prominent J, with complexes slurred and unidentifiable; on January 30, 1954, some complexes were recognizable with J-K complex deeper, but L wave was still as prominent as J in most complexes (Grade 4 to Grade 3).

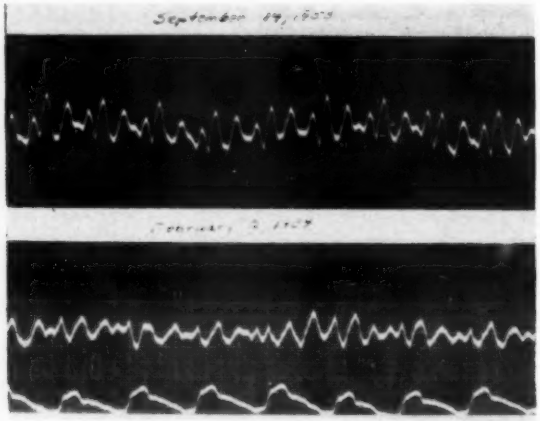


Fig. 11. On September 14, 1953, H-J was fused with L most prominent; complexes were unidentifiable. On February 2, 1954, H-J was still fused and L still more prominent; all complexes were unidentifiable.

class described by Gofman or by alterations of the ratio of alpha-lipoproteins to beta-lipoproteins described by Barr. Both S_f 12-20 and beta-lipoproteins are low in young females.

3. It has been demonstrated that the use of estrogen will favorably alter the alpha-lipoprotein-beta-lipoprotein content of the blood and that estrogen plus androgen will inhibit the production of experimental atherosclerosis.

4. A small group of patients with known coronary artery disease were studied with the ballistocardiograph while they were on a regimen of estrogen plus androgen. The results were indecisive but encouraging, but side-effects were discouraging. It would seem at this time that the ideal preparation would be an estrogen that is nonfeminizing, and that the only other therapeutic modality of value is a low fat diet.

Acknowledgment

Grateful acknowledgment is expressed for the courtesy of the Schering Corporation in supplying Estinyl and to the Upjohn Company in supplying Depo-testosterone. I also wish to acknowledge the kind assistance of Robert Hafer, D.O., and Helen Taleff, R.N., in preparing this material.

122 Muench St.

REFERENCES

1. Oppenheim, F.: Review of 100 autopsies of Shanghai Chinese. China M. J. 39:1067-1076, Dec. 25, 1925.
2. Steiner, P. E.: Necropsies on Okinawans; anatomic and pathologic observations. Arch. Path. 42:359-380, Oct. 1946.
3. Moon, H. D., and Rinehart, J. F.: Histogenesis of coronary arteriosclerosis. Circulation 6:481-488, Oct. 1952.
4. Ackerman, R. F., Dry, T. J., and Edwards, J. E.: Relationship of various factors to degree of coronary atherosclerosis in women. Circulation 1:1345-1354, June 1950.
5. Wilens, S. L.: Bearing of general nutritional state on atherosclerosis. Arch. Int. Med. 79:129-147, Feb. 1947.
6. Smith, H. L.: Incidence of coronary sclerosis among physicians as compared with members of other occupations. J. Am. M. A. 108: 1327-1329, April 17, 1937.
7. Landé, K. E., and Sperry, W. M.: Human atherosclerosis in relation to cholesterol content of blood serum. Arch. Path. 22:301-312, Sept. 1936.
8. Hirsch, E. F., and Weinhouse, S.: Role of lipids in atherosclerosis. Physiol. Rev. 23:185-202, July 1943.
9. Hueper, W. C.: Experimental approaches to problem of arteriosclerosis. Geriatrics 2:293-296, Sept.-Oct. 1947.
10. Steiner, A., Kendall, F. E., and Bevans, M.: Production of arteriosclerosis in dogs by cholesterol and thiouracil feeding. Am. Heart J. 38:34-42, July 1949.
11. Gertler, M. M., Garn, S. M., and White, P. D.: Diet, serum cholesterol and coronary artery disease. Circulation 2:696-704, Nov. 1950.
12. Keyes, A.: Human atherosclerosis and diet. Circulation 5:115-118, Jan. 1952.
13. Pollack, O. J.: Reduction of blood cholesterol in man. Circulation 7:702-706, May 1953.
14. Gofman, J. W., *et al.*: Blood lipids and human atherosclerosis. Circulation 2:161-178, Aug. 1950.
15. Aldersberg, D.: Unpublished report.
16. Wuest, J. H., Jr., Dry, T. J., and Edwards, J. E.: Degree of coronary atherosclerosis in bilaterally oophorectomized women. Circulation 7:801-809, June 1953.
17. Rivin, A. U., and Dimitroff, S. P.: Incidence and severity of atherosclerosis in estrogen-treated males, and in females with hypoestrogenic or hyperestrogenic state. Circulation 9:533-539, April 1954.
18. Barr, D. P.: Some chemical factors in pathogenesis of atherosclerosis. Circulation 8:641-654, Nov. 1953.
19. Stamler, J., Pick, R., and Katz, L. N.: Prevention of coronary atherosclerosis by estrogen-androgen administration in cholesterol-fed chick. Circulation Research 1:94-98, Jan. 1953.
20. Katz, L. N.: Experimental atherosclerosis. Circulation 5:101-114, Jan. 1952.
21. Brown, H. R., Jr., Hoffman, M. J., and deLalla, V., Jr.: Ballistocardiographic findings in patients with symptoms of angina pectoris. Circulation 1:132-140, Jan. 1950.

The Treatment of Acute Nephritis

RICHARD P. DE NISE, D.O.

Chairman of the Division of Medicine
Des Moines Still College of Osteopathy and Surgery
Des Moines, Iowa

The management of acute glomerulonephritis today has been improved because of a clearer insight into the mechanisms of the pathologic processes involved. There are some aspects such as etiology and mechanism of vasoconstriction, that have not yet been fully explained. However, an understanding of the known and explainable changes in the physiologic and pathologic processes will lead to more rational therapy.

The etiology of acute glomerulonephritis has not been satisfactorily explained. Approximately 80 per cent of cases are preceded by a hemolytic streptococcic infection.[1,2] However, the organisms are never found in the kidney. Because of this lack of actual bacterial invasion of the kidney and since the onset of the nephritis is some 10 to 21 days after the streptococcic infection, an antigen-antibody mechanism has been proposed as a causal factor.[2,3,4] If a sensitization process is the etiologic factor, then early antibiotic therapy should be of value in the prevention of the disease process. Weinstein and his associates,[5] however, found that adequate and early penicillin therapy did not reduce the incidence of acute hemorrhagic nephritis. Derow[6] suggests that sensitization in susceptible patients occurs too rapidly for antibiotic therapy to be effective.

The pathologic processes of acute nephritis involve the glomeruli primarily. The arterioles and tubules are not involved until after the loss of the glomerular circulation. There is proliferation and swelling of the visceral capsule, basement membrane, and endothelial cells of the capillaries. The proliferation and swelling are so great as to produce obstruction of the capillary loops. With obstruction of the capillaries, the glomeruli become bloodless, and occupy all of Bowman's capsule;[1,7,8] and some tubular changes then result which, in most cases, are minimal.

Renal blood flow during the acute process is within normal limits. However, the glomerular filtration rate is decreased owing to the primary involvement and obstruction at the glomerular level. The decrease in the glomerular filtration rate allows for an increased reabsorption of salt and water by the tubules, producing oliguria, hypervolemia, and edema. It also has been suggested that the edema is due to a generalized increase in capillary permeability.[9] Edema results in increased venous pressure which in turn may lead to cardiac enlargement and congestive heart failure.[10]

The decrease in filtration rate also causes azotemia and retention of sulfates and phosphates with a resulting mild acidosis.[11] Albuminuria and hematuria apparently are due to the glomerular involvement and capillary permeability. Hypertension is a relatively common finding in acute glomerulonephritis. The exact mechanism by which the hypertension is produced is not known, but it has been suggested that a generalized vasoconstriction is responsible.[11,12] The hypertension that occurs has been shown not to be compensatory in origin because the renal clearance rate and renal plasma flow are not improved by the increased blood pressure.[7] The hyperpiesia may produce a rapid onset of cerebral symptoms due to encephalopathy. That the cerebral involvement is due to hypertension and not azotemia is borne out by the immediate relief obtained by lowering the blood pressure. Also individuals developing signs of encephalopathy do not have sufficient nitrogen retention to account for the severity of the symptoms.

The prognosis for children with the initial attack of acute nephritis is good with the large majority recovering.[6] Statistics vary as to the number of patients who expire during the initial illness from acute kidney failure, congestive heart failure, or hypertensive encephalopathy. A few will progress into the chronic form of glomerulonephritis. Progressive increases in the age of individuals developing acute glomerulonephritis result in a greater incidence in the chronic form.

The treatment of acute glomerulonephritis resolves itself into prophylaxis, management of acute nephritis with no complications, and management of acute nephritis with complications. For the most part, therapy is relatively simple if the basic physiologic principles are kept in mind, particularly that the greatest number of patients recover, many without the aid of any specific therapeutic measures.

It would appear that if the etiologic factor is a hemolytic streptococcus then antibiotic therapy administered during the acute infection should eliminate the appearance of glomerulonephritis at a later date. Fishberg[13] states that although many cases of glomerulonephritis have occurred following streptococcic infections, he believes the incidence of the disease has decreased in New York City since the advent of antibiotics. Weinstein and his associates,[5] in a study of 167 patients, found that early penicillin therapy in the treatment of streptococcic infections did not prevent the occurrence of glomerulonephritis. Penicillin therapy did, however, definitely decrease the severity of the clinical course of acute nephritis. It would seem that antibiotic therapy has a definite part in the prophylactic treatment. On the other hand, antibiotics are not indicated in the management of nephritis itself unless they are used to overcome or prevent secondary infectious processes.

Since an antigen-antibody reaction has been suggested as one of the mechanisms resulting in acute nephritis, antihistamine therapy has been utilized to treat this disease process. Early reports concerning the use of antihistaminic therapy were somewhat favorable.[14] However, the earlier results have not been borne out by further investigation.[6,13] Based on the same premise of an allergic origin of the pathologic process, ACTH and cortisone have been used in the management of acute nephritis. Clinical investigation of this type of therapy has shown no remarkable results.[15,16] As a matter of fact, if anuria or oliguria and edema are present, ACTH and cortisone therapy could increase the retention of salt and water and thus intensify an already dangerous situation.

Since the greatest number of patients with acute nephritis develop few marked symptoms, therapy for the most part is limited to rest and diet and fluid regulation. All patients suffering from this disease process, regardless of severity, should have absolute bedrest en-

forced. Renal blood flow is increased in the recumbent position.[10] The length of time which the patient should remain in bed will depend upon the individual's reaction to the disease process. Ideally, bedrest should continue until all edema and hypertension have disappeared and until all red cells and protein have disappeared from the urine. Many patients continue to show some urine abnormalities after all other symptoms have disappeared, and it is maintained that this indicates the disease process is still active. Authorities expressing this viewpoint believe the patient should remain in bed until the urine is absolutely normal or for at least 6 months.[17] The remainder feel that bedrest should continue only until all other clinical signs of activity, such as hypertension, edema, and azotemia, have disappeared. The return of the individual to normal activity should be gradual and governed by repeated examinations of urine.

The diet and fluid therapy prescribed will depend upon the urinary output and the degree of azotemia, edema, and hypertension. In those instances in which the urinary involvement is slight there will be no need to impose strict dietary and fluid regimens.[18] The individuals who develop any great degree of azotemia, however, should be placed on a high calorie, high carbohydrate, and high fat diet. Protein intake should be no greater than 20 grams in any 24-hour period. The high calorie, high carbohydrate, and high fat diet will slow protein catabolism and the production of nitrogenous waste products.[13,17] The individual exhibiting azotemia, generally speaking, also has some oliguria with sodium and potassium retention. Retention of these electrolytes will lead to the development of edema, hypertension, and congestive heart failure; therefore, the diet should contain less than 1 gram of salt per day and should be low in potassium.

The regulation of fluid intake will depend upon the urinary output. If there is no oliguria, then there need be no restriction. The patient, when able to take fluids orally, will automatically regulate the intake-output ratio. However, if nausea and vomiting or coma is present, fluids must be given parenterally. The day of pushing fluids to "flush the kidneys" is past. Parenteral fluid intake should be limited to the urinary output for the past 24 hours plus insensible fluid loss.[19] The fluid should be given in the form of glucose in water, unless nausea, vomiting, and diarrhea have been present, in which case there may be sodium, chloride, and potassium deficiencies. Daily electrolyte studies should be performed and electrolyte replacement governed by the results of the laboratory studies. In those cases in which anuria or severe oliguria develops, the use of the artificial kidney and peritoneal and intestinal lavage have been recommended. Fishberg[13] maintains, however, that results with conservative management consisting of diet, fluids, and electrolytes compare favorably or are better than results with the use of peritoneal and intestinal lavage and the artificial kidney.

When hypertensive encephalopathy occurs, immediate therapy is indicated. During this period the blood pressure is very high, apparently because of generalized vasospasm. Magnesium sulfate, both clinically and experimentally, has reduced the elevated blood pressure. Harris and De Maria[12] administered magnesium sulfate to a group of patients with acute nephritis and found that there was a marked increase in the glomerular filtration rate and the renal plasma flow and a prompt decrease in blood pressure. In a group of normal children no change in the glomerular filtration rate, the renal plasma flow, or blood pressure was noted. The oral dosage of magnesium sulfate is from ½ to 1 ounce of saturated solution three to four times daily. Many patients are unable to take the medication orally, and then it may be administered intramuscularly in a 25 per cent solution at the rate of .4 cc. per kilogram of body weight every 4 hours until results are obtained. The substance may also be given intravenously as a 2 per cent solution in 5 per cent glucose at the rate of 40 to 50 cc. per hour.[2] The blood pressure should be checked frequently during both intramuscular and intravenous administration and the dosage adjusted to coincide with changes in pressure. Light magnesium carbonate has been suggested as a substitute for magnesium sulfate because it is less nauseating and tastes better. To obtain a therapeutic effect it is necessary to produce four to six diarrheic stools per day. The recommended dosage is 2 tablespoons every 4 hours depending upon results.[20] Use of this drug, however, is not intended to replace parenteral magnesium sulfate.

Symptoms of encephalopathy may be an indication for the use of the newer hypotensive drugs. Although there has been little written of their use in hypertensive encephalopathy due to acute glomerulonephritis, the rationale for their use is present. All of these new products produce a generalized vasodilatation, although the mechanism by which the effect is obtained varies with the drug used.[21,22] In hypertensive emergencies, the parenteral administration produces the best results. Intramuscular or intravenous use of hexamethonium, hydralazine, or Veratrum will be the most effective in alleviating the signs and symptoms of encephalopathy and lowering the blood pressure.[23] Parenteral administration requires careful and extremely frequent blood pressure evaluations with changes in the rate of administration coinciding with the depression and elevation of the systemic pressure. It has been my experience with the use of hexamethonium that shock levels of the blood pressure can occur very rapidly and with extremely small amounts of the drug. If extreme hypotensive levels do occur they can be counteracted by the administration of vasoconstricting substances.

In encephalopathy, the spinal fluid pressure is elevated because of cerebral edema. Spinal tap with reduction in pressure may improve the symptoms of encephalopathy; however, the inherent dangers with this procedure outweigh the advantages and the method should probably be limited to extreme emergencies.[23]

The remaining complication, congestive heart failure, is managed in the usual manner with digitalis and oxygen, and, if it is acute in onset, positive pressure oxygen, and alcohol vapor inhalation may be of value.

SUMMARY

The etiology, altered physiology, and therapy of acute glomerulonephritis have been discussed. The mechanisms of etiology, vasoconstriction, and hypertension have not been satisfactorily explained to date.

The general therapeutic measures have been mentioned, and the use of the newer hypotensive drugs has been suggested.

726 Sixth Ave.

REFERENCES

1. Boyd, W.: Pathology of internal diseases. Ed. 5. Lea & Febiger, Philadelphia, 1950.

2. Grulee, C. G., and Eley, R. C.: The child in health and disease. Ed. 2. Williams & Wilkins Co., Baltimore, 1952.

3. Lange, K., *et al.*: Autoantibodies in different phases of human glomerulonephritis. Bull. New York Acad. Med. 25:447, July 1949.
4. Addis, T.: Glomerular nephritis. Macmillan Co., New York, 1948.
5. Weinstein, L., Bachrach, L., and Boyer, N. H.: Observations on development of rheumatic fever and glomerulonephritis in cases of scarlet fever treated with penicillin. New England J. Med. 242:1002-1010, June 29, 1950.
6. Derow, H. A.: Management of acute glomerulonephritis. New England J. Med. 249:144-153, July 23, 1953.
7. Smith, H. W.: The kidney. Oxford University Press, New York, 1951.
8. McManus, J. F. A.: Medical diseases of kidney. Lea & Febiger, Philadelphia, 1950.
9. Bradley, S. E.: Discussion (p. 382) on Acute diffuse glomerulonephritis. Combined Staff Clinics, Am. J. Med. 7:382-395, Sept. 1949.
10. Hall, A. D., and Luetscher, J. A., Jr.: Renal disease. New England J. Med. 239:621-631, Oct. 21, 1948.
11. Harrison, T. R., ed.: Principles of internal medicine. Ed. 2. Blakiston Co., New York, 1954.
12. Harris, J. S., and DeMaria, W. J. A.: Effect of magnesium sulfate on renal dynamics in acute glomerulonephritis in children. Pediatrics 11:191-205, March 1953.
13. Fishberg, A. M.: Bedside management of acute glomerulonephritis and nephrotic syndrome. M. Clin. North America 37:1129-1137, July 1953.
14. Craig, J., Clark, N. S., and Chalmers, J. D.: Antihistamine drug treatment of acute nephritis. Brit. M. J. 1:6-9, Jan. 1, 1949.
15. Burnett, C. H., *et al.*: Effects of cortisone on course of acute glomerulonephritis; report of case. New England J. Med. 243:1028-1032, Dec. 28, 1950.
16. Thorn, G. W., *et al.*: Clinical studies with ACTH and cortisone in renal disease. Arch. Int. Med. 86:319-354, Sept. 1950.
17. Duncan, G. G., ed.: Diseases of metabolism. Ed. 3. W. B. Saunders Co., Philadelphia, 1952.
18. Burke, E. C.: Fluid regulation in nephritis and nephrosis of childhood. Collect. Papers Mayo Clin. & Mayo Found. 45:267-273, 1953.
19. Bland, J. H.: Clinical use of fluid and electrolyte. W. B. Saunders Co., Philadelphia, 1952.
20. Thenebe, C. L.: Light magnesium carbonate in treatment of acute glomerular nephritis; preliminary report. Arch. Pediat. 69:285-287, July 1952.
21. Livesay, W. R., Moyer, J. H., and Miller, S. I.: Treatment of hypertension with Rauwolfia serpentina alone and combined with other drugs; results in eighty-four cases. J. Am. M. A. 155:1027-1035, July 17, 1954.
22. Stunkard, A., Wertheimer, L., and Redisch, W.: Studies on hydralazine; evidence for peripheral site of action. J. Clin. Investigation 33:1047-1053, July 1954.
23. Hopper, J., Jr., Bolomey, A., and Wennesland, R.: Chronic renal insufficiency. I. Appraisal of patient. Ann. Int. Med. 41:18-35, July 1954.

Clinical Observations on the Herniated Intervertebral Lumbar Disk

JAMES M. EATON, D.O., F.A.C.O.S.
Professor of Orthopedic Surgery
Philadelphia College of Osteopathy
Philadelphia

Historically the finding of certain cartilaginous masses within the spinal canal and the epidural spaces, the annulus fibrosus and the nucleus pulposus of the intervertebral disk, dates back to the work of Schmorl[1,2] who called attention to it in 1928 and again in 1929.

However, it was not until 5 years later that Mixter and Barr[3] pointed out the clinical significance of these findings and demonstrated that root compression and irritation resulting from hernias of the annulus fibrosus and nucleus pulposus were etiologic factors in low-back and sciatic pain.

Since that time there have been innumerable references to this pathologic entity in the literature, and although the subject has been thoroughly discussed and investigated over the past 20 years, the controversy still rages and differences of opinion with regard to it and to methods of treatment become very heated at times.

The diagnosis is not easy and will tax the ability and ingenuity of the examining physician. There are those who claim that any low-back complaint with or without sciatic pain is due to a rupture of one or more of the lower lumbar intervertebral disks, and they further claim that diagnosis can be made only on the operating table. There are others who claim that surgery should be performed only when there is incontrovertible proof of the presence of such lesions, and still others say that surgery should be performed only after prolonged conservative management has failed.

A careful history and physical examination of the patient supplemented by roentgenologic studies of the lumbar spine and pelvis and the ability to evaluate and interpret the findings from such laboratory studies are most essential. The ability of the clinician to evaluate his patient as an individual and to interpret his clinical findings and the ability of the radiologist to interpret his studies are of utmost importance, but not all possess the acumen necessary to perform these examinations to the complete satisfaction of the attending physician or themselves.

Much has been written about the typical history of the so-called disk syndrome, but from my own experience there is no such thing as a typical disk syndrome. The pain and disability which is produced by such nerve root irritation is no more characteristic of a rupture of one or more of the lower lumbar intervertebral disks than it is characteristic of the arthrodial lesion of Still, of primary and secondary malignancies of the spine, or of reflex irritability of viscerosomatic origin.

The ruptured intervertebral disk will produce pain in two general locations, or, rather, will result in two pain syndromes. With rupture or protrusion of the annulus fibrosus in the midline or laterally without causing irritability of the nerve root itself, there will frequently be caused only low-back pain due to the irritating effects of the annulus on the sensory fibers which lie in the posterior common ligament. This will produce low-back pain of a rather vague nature which often is treated for many months or even years by one means or another with variable results, depending upon the ability of the tissues in the area involved to control the herniated annulus fibrosus and to prevent it from becoming of major significance. If the ligamentary structure is strong enough to hold the annulus fibrosus within reasonable bounds, low-back pain will exist but may be entirely eliminated as the irritability of the sensory fibers in the posterior common ligament either becomes relieved or exhausted.

After months or even years of low-back pain, in many cases the pain suddenly shifts from the lower back to involve branches of the sciatic nerve and may affect the sciatic nerve and its branches almost anywhere along its distribution, depending upon the nerve root involved. In analyzing this type of syndrome, the herniation of the annulus fibrosus, which formerly was in the midline or slightly lateral, but not enough to produce irritability of the nerve root, now suddenly bursts through its retaining bond of the posterior common ligament and extends laterally to engage the nerve root and cause either irritability or actual compression of the nerve root structure within the spinal canal close to the intervertebral foramen, thus producing the dra-

matic sciatic pain of which so many of these patients complain.

If any of these patients presents a so-called typical syndrome it is nothing more than the syndrome of radicular pain, that is, irritability of the nerve root structure itself in a patient who otherwise does not present clinical or radiographic findings sufficient to produce such radicular pain.

Unfortunately, in lesions of the lumbar disks, the neurologic signs of irritability of the lumbar nerve roots are not clearly defined or demonstrated, and there is a paucity of neurologic signs or symptoms in the ruptured intervertebral disk.

In evaluating the low-back complaint of the patient the physical examination is conducted in an attempt to differentiate between muscular and ligamentous lesions, arthrodial lesions, involvement of the nerve root structures themselves, the so-called radicular signs, and painful conditions of the sciatic nerve after it has emerged from the sacrosciatic notch to the periphery.

Some years ago an intensive study[4] of the low back with reference to this painful syndrome was conducted. In it I described the great variety of signs which I felt were necessary and of value in establishing a sound basis for clinical diagnosis. Those physical signs which are indicative of nerve root irritability are highly suggestive, but one cannot depend on any one sign or finding, or for that matter on a set pattern of physical signs, to establish a diagnosis in any of these cases. The patient must be studied as an entity, and after all the facts have been gathered they may then be evaluated and conclusions reached.

After clinical evaluation of the patient and a presumptive location of the cause, x-ray studies of the lumbar spine and pelvis should be carried out as a means of determining the presence of inflammatory, degenerative, neoplastic, or traumatic involvement of the lumbar region and its relationship to the pelvis. This is most important because concomitant involvement of the spine such as chronic spondylarthrosis or spondylarthritis will influence the result from surgery or any other form of therapy that is employed, and it is well to acquaint the patient with these facts before proceeding with further examination or therapy. Frank discussion with the patient and his family of the various problems presented will avoid uncomfortable criticism during the postoperative period.

Special x-ray technics have been employed to aid the clinician in establishing the diagnosis of ruptured intervertebral disk, all of which have their advantages and disadvantages. They are all expensive and should not be a part of a routine procedure, but rather these special technics should be confined to those cases which present definite signs of nerve root irritability and those in which adequate conservative osteopathic manipulative therapy has failed to give the patient the relief that he should obtain from such therapy.

Oil myelography employing either Lipiodol or Pantopaque will be disappointing in many cases because, if the spinal canal is quite large and the dural sheath comparatively small, the oil will not demonstrate extremely laterally placed disk herniations unless the dura can be forced against the herniation and produce a visible defect. However, oil myelography will be of extreme help in those cases in which it is essential to differentiate between disk herniation and tumor elsewhere in the lumbar or lumbodorsal column, since other diagnostic procedures would fail to demonstrate such lesions. The procedure itself is not too difficult, but unless the oil is removed immediately, and this requires painstaking technic, some of it will be deposited in the nerve sheaths and may be a source of irritation for some weeks or months. I have not seen any permanent ill effects from such myelography, but it is potentially harmful.

I have had considerable experience with air myelography, having use it rather extensively for quite some time. The one great problem with air myelography is that it requires a radiologist who is exceedingly skilled in interpreting the findings. Its big advantage is that after the air is absorbed there cannot be any irritating substances left behind which would cause trouble at a later date. Air myelography has another advantage over oil myelography in that the dural sac can be ballooned laterally, and thus interpreted, defects lateral to the normal excursion of the dura can be seen, and a greater number of positive findings are provided than with oil myelography.

Postspinal headache occurs with both of these technics, I believe slightly more frequently with air myelography. However, if precautions are taken after the procedure is completed this complication is not too objectionable.

Since 1950 we have been employing at the Hospital of the Philadelphia College of Osteopathy a technic known as Diodrast nucleus opacification. This technic, in contrast to oil myelography or air myelography, gives a positive demonstration of disk herniation because if the continuity of the annulus is broken the radiopaque material escapes into the epidural space and can be demonstrated on the x-ray films.

The technic requires very close collaboration between the radiologist and the surgeon, but when properly performed will provide diagnostic information relative to the integrity of the lumbar intervertebral disks which is most positive and accurate. In addition, the findings are such that disk herniation can be proved irrespective of whether the herniation is posterior, lateral, or anterior.

In brief, the technic consists of introducing a needle directly into the area of the nucleus pulposus and of checking its position by anterior and posterior radiographs to make certain that it is placed as accurately as possible directly to the centrum of the nucleus pulposus. Injection of 1 to 2 cc. of 35 per cent Diodrast is made with the patient in the prone position. Anteroposterior and lateral studies are then made which reveal whether the radiopaque substance has remained within the annulus fibrosus or if it has escaped and in what direction, thus giving visual proof of the loss of integrity of the annulus fibrosus. In certain cases Diodrast escapes through the annulus fibrosus but is still retained by the posterior common ligament. This is the so-called retropulsed or protruding disk herniation and can be determined with this technic.

After a rather extensive experience with this procedure I am impressed with its accuracy in determining herniation of the lumbar intervertebral disk and also by the fact that many times when the disk is exposed at surgery the herniation is not apparent to the eye. If this is not removed at surgery the patient may well have a recurrence of his lesion at a later date. I feel that a great many of the patients, by virtue of this technic, have escaped disk herniation postoperatively.

At surgery the findings have been extremely accurate, both from the standpoint of the herniation and also from the standpoint of the normal radiographic

findings. Not a single case has been observed in which herniation diagnosed from the x-ray study was not encountered at the time of surgery. The reverse has also been true: The intact disk at x-ray study has been found to have normal appearance and consistency at surgery.

The one disadvantage of the technic is that unsuspected tumors of the cauda equina or of the lumbodorsal region will not be observed; however, if the multiplicity of laboratory studies was carried out which might be considered necessary to rule out the possibility of all diagnostic potentialities, the patient might well be exhausted both metabolically and economically.

One sometimes hears the opinion that needle holes placed in the annulus fibrosus are a potential cause of disk herniation in an otherwise undamaged disk. I have searched for evidence of such defects at the time of surgery. Thus far I have been unable to discover any defect which can be attributed to the procedure, even when the annulus is obviously damaged and degenerated. It is my opinion that the annulus is self-sealing to such needle punctures.

The findings at surgery are not always compatible with the physical findings or the history. Extremely large massive herniations have been found which produced very meager physical findings, though the history of the case might indicate that a major involvement did exist.

Consideration must be given to the various anomalies of development which take place throughout the human body, particularly in the lumbosacral area which probably has more than any other part of the body. An extremely small disk herniation discovered at surgery may be of major significance if it is in an area where the spinal canal for some reason is decreased in diameter or size and thus reduces the possible excursion of the nerve root. The radiologist on many occasions has demonstrated the extremely short pedicle, the anomalies of development of the articular processes and the laminae. The thickness of the ligamentum subflavum also plays an important part in irritating the epidural space in many instances. Then, too, there are many anomalies in the way nerve roots emerge from the dural sheath which obviously influence their degree of irritability. Many of these disk herniations occupy an extremely lateral position and project directly into the intervertebral foramen and irritate the nerve root in this location where it has little or no chance to escape such irritation.

It has been fairly well established that disk herniations occur principally at the transitional levels of the spine, namely, the lower cervical and the lumbosacral regions. The disk herniation occurs in those areas of the spine where the normal lordosis is at its greatest significance, and from the natural forces of gravity in such locations there is bound to be a greater degree of stress upon the posterior annulus than at any other point. Hence, the normal forces of the erect position of the human body would tend to predispose to weaknesses in the annulus from the wear and tear of everyday life.

Coupled with this is the fact that practically every one of us at some time in his life has experienced an injury in which acute flexions of the spine have occurred either in the standing or the sitting positions. Such injuries naturally produce the greatest stress and strain in the areas of greatest mobility and in the areas of greatest change in the anteroposterior curves of the spine.

I have also observed that in most of these cases anatomic peculiarities within the spinal canal have been suggestive. The posterior common ligament is not always the well-developed structure that it is believed to be. Deficiency in the actual content of the posterior common ligament is encountered in most instances where disk herniation has been demonstrated. Normally the posterior common ligament is strongest in the midline. It is as it fans out to either side to give support to the annulus in its lateral projections that the greatest variety of anomalies is found. Sometimes the ligament itself appears to be entirely absent, while in other cases it is so thin that it almost appears to be on the verge of spontaneous eruption, the tension beneath it from the ruptured disk being so great. Of course, there are the obvious cases in which the nucleus pulposus has ruptured through both the annulus and the posterior common ligament with the escape of pulposus material within the spinal canal, and in such cases again I have observed deficiencies in the development of the posterior common ligament.

I also have noticed that in the great majority of cases of herniation, when it is of the so-called subligamentary type, that the nucleus pulposus seems to protrude between the annulus fibrosus and the body of the vertebra as if it became detached for one reason or another and allowed for a gradual seepage of the soft nucleus pulposus between the annulus and the body of the vertebra. By far the great majority of these escape between the inferior border of the annulus and the superior border of the vertebral body below, irrespective of the level involved. This might suggest a developmental anomaly within the disk itself. Then again, the anteroposterior diameter of the posterior portion of the annulus fibrosus is much thinner than elsewhere in the disk itself.

With the multiplicity of possibilities of weakened anatomic structures that may exist, coupled with a strain mechanism with the patient in an anteflexed attitude combined with torsion, it is not too difficult to understand the forces at play when the disk finally ruptures in a posterolateral position.

It is also an interesting fact that cartilage has no demonstrable blood supply or sensory nerve innervation. Nutrition is gained through the synovial fluid and to a slight extent through the subchondral bone to which it is loosely attached. Elasticity of cartilage is exhibited over short periods, and, as the period or duration of deformation increases, the elasticity of the affected area decreases. Prolonged deformation of cartilage may result in the death of the chondrocytes as well as in permanent deformity. In addition, elasticity of cartilage normally decreases with the inspissation of age.

The annulus of the intervertebral disk is fibrocartilage in contrast to hyaline cartilage found elsewhere. Fibrocartilage has a much higher percentage of collagenous fibers with a decrease in the amount of matrix and the number of chondrocytes; hence, it has a much higher tensile strength than hyaline cartilage.

Damaged cartilage of either type, that is, hyaline cartilage or fibrocartilage, does not possess the power of repair to any degree, since cartilage which has undergone dehiscence does not reunite. Local proliferation may occur to a certain degree, but no side-to-side union may be expected in such traumatic defects.

Degeneration of the intervertebral disks is a serious problem. Dead tissue anywhere in the living body is capable of producing pain. In addition to the collapse of the disk caused by weight bearing on such diseased structures, arthrodial changes are produced which in themselves are capable of producing radicular signs as serious as those from a ruptured intervertebral disk.

Narrowing of the disk interspaces by degeneration of the disk causes a settling of the inferior facets on the superior facets. Rotary motion is increased because of relaxation of the annular and longitudinal ligaments. Contracture of the supporting ligaments is an attempt to stabilize the area. Pain will result from the increase of ligamentous strain and from the efforts of intrinsic muscles to stabilize the articulation. The superior structure may also move posteriorly and produce a so-called reverse spondylolisthesis.

With advancing age there is gradual degeneration in the annulus fibrosus which produces mechanical weakness. At the same time the nucleus pulposus gradually becomes inspissated.

From all that I have been able to determine from studies of the intervertebral disks the changes which take place in cartilage are irreversible.

It is the responsibility of the general practitioner to acquaint his patient with the presumptive diagnosis of disk herniation and adequately prepare him for the possibility of radical surgical treatment. He thus protects himself against losing the confidence of his patient if conservative treatment fails, and, what is more important, gives the patient a chance to prepare himself emotionally and economically for the necessary surgery.

Prolonged conservative treatment to the point of economic and nervous exhaustion doubles the hardship to the patient by creating an additional emotional problem which is sometimes more difficult to eradicate than disk herniation. Disabling back pain is a personal, social, and economic disaster difficult for the attending physician to appreciate unless he himself has been subject to this syndrome.

It is most important that the physician train himself to observe and note the changes in the central nervous system indicating impending damage to the nerve roots and to place these facts before his patient and to advise consultation and thorough study to determine the site and cause of his disability before it is too late. When weakness has occurred in the musculature it is usually too late to rehabilitate these patients completely. When symptoms from cauda equina involvement have developed, recovery has never been complete, nor has recovery from muscle paralysis.

Disk herniation in itself does not make a man unable to work; it is the associated pain and disability which incapacitates him.

The dangers surrounding prolonged conservative management of a patient suffering from herniated lumbar intervertebral disk are many:

1. Progressive degeneration of nerve root structures may exist and remain in a subclinical phase for long periods and be undiagnosed until they are full blown and irreversible, at which time it is too late to expect satisfactory results from surgery.

2. A false sense of security exists when pain recedes, even though weakness, paresthesias, and altered reflexes persist. These are definite signs of permanent changes in the central nervous system which must be guarded against.

3. A false sense of security will also exist in those cases in which acute spasmodic pain subsides but low grade pain persists. This low grade pain can be tolerated by the patient and may be considered as an improvement. In these cases the nerve root irritation persists in spite of the lessening pain.

4. Whenever these conditions exist, good clinical results cannot be expected, complete recovery rarely occurs, and the residual syndrome develops much to the dissatisfaction of all concerned.

5. Complicating degenerative and mechanical changes take place in the osseomusculoligamentous systems which will offset the results of surgery and require prolonged expensive rehabilitation of the patient.

6. Diminution or loss of reflexes may indicate degeneration or exhaustion of nerve root irritability, the patient thus being incapable of responding to stimuli. There is no test to differentiate accurately between exhaustion and degeneration, and one must act promptly to preserve that which remains.

7. It is important to operate before fibrous adhesions from inflammatory reaction develop in and about the nerve root structures. The results are not good in these cases because of the underlying inflammatory and degenerative root changes.

The limitations of the surgical approach must be kept in mind in the management of these cases. Success of operation is in direct proportion to the permanent degenerative and irreversible changes that have taken place in the nerve root structures. Surgery can only remove the source of the nerve root irritation.

In my opinion the following are indications for surgical intervention in a patient suffering from a rupture of one or more of the lower lumbar intervertebral disks.

1. Every case in which there is a suspicion that the cause of nerve root irritation is a rupture of one or more of the lower lumbar intervertebral disks should be considered as a potential surgical problem.

2. Failure of the patient to respond to adequate conservative management, even when clinical signs of nerve root damage are meager.

3. Demonstrable extradural lesions on myelography or positive signs of disk herniation by Diodrast nucleus opacification not only establish the diagnosis but are a direct indication for surgical intervention.

4. Degeneration of the intervertebral disk with or without demonstrable herniation, where the symptomatology is recurrent and fails to respond to adequate conservative management.

5. Radicular signs with a disk syndrome in the absence of other evidence of disease of the central nervous system.

6. Radicular signs in the presence of arthrodial damage or other pathologic conditions.

7. Those cases in which radicular pain predominates or in which the patient has given a history of recurrent low-back pain but recently has exhibited an increase and preponderance of radicular pain.

It is my considered opinion that the majority of patients who suffer from a so-called disk syndrome also suffer from an instability of the lumbosacral region which if not recognized and stabilized at the time of surgery will influence the results of any surgical technic. Whether or not lumbosacral instability antedates the disk syndrome is not important, but it is extremely important to recognize its existence and to correct it if possible.

It has been my practice for some years to stabilize all backs where disk herniation has been discovered and

treated surgically. Comparative results in a large number of cases have indicated that the postoperative result as expressed by the patient has been far better when spinal fusion was a part of the treatment of the low-back syndrome than when only the herniated disk was removed.

Operation for ruptured intervertebral disk should not be performed on those patients who respond to adequate conservative management in whom there is no demonstrable evidence of organic change in the nerve root structures and on patients who, because of various physical or metabolic difficulties, are considered poor surgical risks.

From the foregoing discussion it may seem that I am almost radical in my approach to the ruptured intervertebral disk problem in the lumbar spine. It has always been my belief that everything possible should be done to establish the diagnosis. Once the diagnosis has been established, the pattern of treatment will unfold itself and become specific. I have followed this program in my cases and have established the diagnosis before recommending surgery, and I do not recommend surgical intervention in any patient unless there is clinical and radiographic confirmation of the diagnosis. However, once the diagnosis has been established I have adhered to the axiom, "Once a disk, always a disk," and, in this paper, have tried to elaborate upon my reasons for this belief.

12 S. 12th St.

REFERENCES

1. Schmorl, G.: Die pathologische Anatomie der Wirbelsäule. Verhandl. d. deutsch. orthop. Gesellsch., Kong 21, pp. 3-41, 1927.
2. Schmorl, G.: Zur pathologischen Anatomie der Wirbelsäule. Klin. Wchnschr. 8:1243-1249, July 2, 1929.
3. Mixter, W. J., and Barr, J. S.: Rupture of intervertebral disc with involvement of spinal canal. New England J. Med. 211:210-215, Aug. 2, 1934.
4. Eaton, J. M.: Symposium: Differential diagnosis and treatment of herniated or protruding intervertebral disk; low-back problem. J. Am. Osteop. A. 46:514-521, June 1947.

Diagnosis and Treatment of Cancer of the Colon

ARTHUR M. FLACK, JR., D.O., F.A.C.O.S.
Clinical Professor of Surgery
Philadelphia College of Osteopathy
Philadelphia

INTRODUCTION

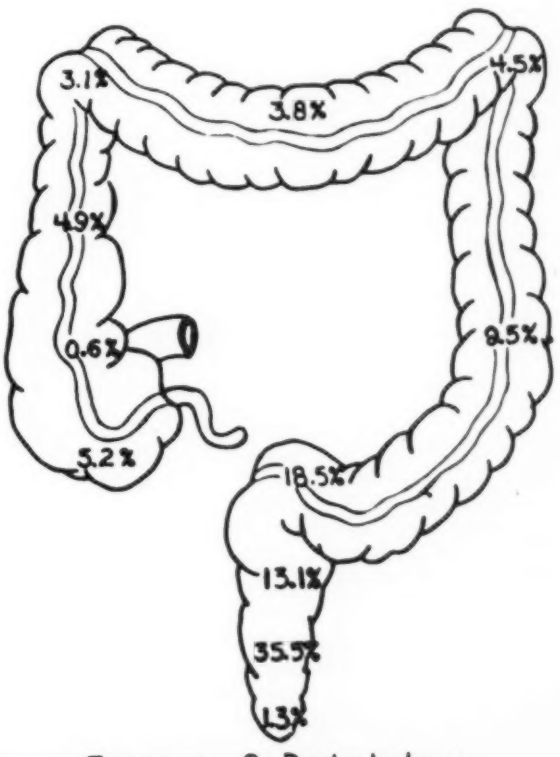
Epithelial malignant disease of the large bowel is a condition acquired by an increasing number of patients of all ages. It is an extremely common condition in both sexes, occurring with about equal frequency in each and constituting about 13 to 15 per cent of all cancer. Fortunately, in the majority of instances, it is relatively slow to produce distant metastases and by the surgical procedures now available that entail a reasonable morbidity and mortality, many cases of the disease can be cured.

Basically, this condition should be found or suspected by the general practitioner, and although its management is in the province of the surgical specialist, he is not the one to depend upon for early diagnosis. Cancer education of the public will undoubtedly produce the best results. As the advice to women relative to regular systematic self-examination of their mammary glands has brought an increasing number of patients with stage I lesions to the physician much earlier for proper treatment, so will continuous reiteration bring early to the physician those individuals with changes in bowel habits or with blood in the stool. Unfortunately, lay education too often is more effective than is the education of the busy physician, who can readily attribute all bleeding at defecation to the existence of an anal fissure or hemorrhoids when these entities are discovered. Until some simple, inexpensive, accurate hematologic or serologic test for cancer of the colon is discovered the best means of diagnosis are simple periodic examinations of the asymptomatic patient and complete evaluation of the patient with changes in habitual bowel function, either constipation or diarrhea, with rectal bleeding, and with anemia. Patients are beginning to understand this latter necessity, through the efforts of the American Cancer Society, and are coming to the physician sooner, demanding routine diagnostic methods instead of resorting to self-medication.

Of all sites for the location of this disease, about 70 per cent are within reach of the examining finger or the sigmoidoscope.

DIAGNOSIS

That lesions of a malignant nature coexist with benign lesions that bleed is well known, and no therapy for the benign condition should be started until the



FREQUENCY OF DISTRIBUTION

presence of a malignant one has been ruled out by complete evaluation.

For the asymptomatic patient subjected to routine checkup, a definite order should be used in the approach to the problem, but the examination does not have to be as extensive and expensive as it should be for those with symptoms. After a routine abdominal evaluation, the basic steps are first, a slow, gentle, and thorough digital inspection of the entire circumference of the anal canal and rectum with the index finger covered with a well-lubricated finger cot.

Steps in the examination of the asymptomatic patient.—

1. Careful, gentle abdominal palpation, beginning over the cecum
2. Digital examination
3. Determination of occult blood in the stool (guaiac test)
4. Proctoscopy
5. Sigmoidoscopy

The steps should always be carried out in the order outlined above.

Two specific items should be mentioned in relation to the manner in which the digital examination is carried out. In the female, it should follow every bimanual abdominovaginal examination, and therefore is done while the patient is still in the conventional lithotomy or supine position with the legs in crutches or feet in stirrups. This procedure supplementing pelvic examination in the female materially adds to the accuracy of the findings gleaned by vaginal examination, owing to the greater depth the examining finger may reach behind the cervix uteri and broad ligaments.

It is to be recommended that the same position also be utilized in the male where possible, in preference to the Sims' or the knee-chest position. In both sexes, the pelvic contents drop deeper into the pelvis in this position, and by the exertion of a moderate degree of intra-abdominal pressure, a greater portion of the distal large bowel comes within reach of the examining finger. The prostate gland and seminal vesicles are simultaneously evaluated in the male with as much ease as in more frequently used positions. The manner of digital insertion is of great importance, especially in relaxing the patient. The ball of the volar surface of the finger is placed against the anal verge and allowed by gentle sustained pressure to enter the anal canal and rectum slowly to the full length of the finger. A little time taken with this step of the examination is appreciated by the patient and materially lessens his hesitancy regarding subsequent re-examinations. For the patient with a tight or spastic sphincter, removal and reinsertion of the finger two or three times prior to circumferential exploration will do a great deal to relax the patient and make the rest of the examination easier.

A screening test for occult blood in the stool is carried out at the completion of the digital examination. The specimen is obtained by wiping the surface of the finger cot onto a paper towel or piece of filter paper immediately after withdrawal of the finger.

Screening Test for Occult Blood in Stool.—

Materials:

1. Saturated solution of guaiacum
2. Glacial acetic acid
3. Hydrogen Peroxide (3 per cent)

Note—Solutions should be kept in dark dropper bottles and tested frequently for potency.

Technic:

1. Smear specimen of stool from examining finger on filter paper.
2. Place 1 to 2 drops of each solution in sequence (as listed above) on the filter paper.
3. Read results in 30 seconds.

Results:

1. Blue or dark green in 30 seconds—positive.
2. Any other color change or delayed color change—negative.

This test is less sensitive than is the benzidrine test and usually does not require a meat-free diet. However, if a positive result is obtained, re-examination is required after 3 days' abstinence from meat.

Proctoscopic evaluation may be done subsequent to the previously mentioned procedures, utilizing for this purpose one of the many specula that are available. Again, gentleness and thoroughness with complete inspection of all portions of the rectal mucosa are of prime importance. Small soft polyps, single or multiple, may be discovered that have not been found by the examining finger. These lesions, as well as polypoid adenoma, constitute a precancerous state and should be removed in their entirety, including the base of the pedicle, for histologic study. For this purpose, one may use either a biopsy forceps as that devised by Yeomans, a cold nasal snare, or an electrocoagulating snare. Care must be exercised that no more than the mucosa of the colonic or rectal wall is removed in order to prevent immediate or delayed perforation. The pathologist is requested to pay particular attention to the cellular characteristics of the pedicle and the possible existence in this location of anaplastic cells. If the procedure is used utilizing an electrocoagulating snare of biopsy loop, care must be taken to provide adequate ventilation of the lumen of the bowel and to evacuate intestinal gases prior to use of the instrument in order to prevent explosion. Fatalities have occurred in instances in which these precautions have not been exercised. Explosions of hydrogen sulfide-oxygen mixtures have resulted in multiple large lacerations locally and higher in the colon with the immediate development of profound shock, and death before laparotomy could be performed to repair the defects.

In the asymptomatic patient reporting for a routine physical examination, sigmoidoscopy is mandatory only at the first examination unless abnormalities are discovered which require re-evaluation. Before this procedure can be performed the distal large bowel must be free of content. This may be accomplished by having the patient take a low cleansing enema the night before and in the morning of the day of examination, using but a pint of warm tap water. There need be no interruption of the usual diet. For the individual who appears for evaluation without this preparation, suitable cleansing can be accomplished within 5 minutes in the office by using one of the phosphate-enema preparations (Fleet Enema or Clyserol).

Many endoscopes have been devised by various manufacturers. They are relatively inexpensive and should be as much a part of the general practitioner's office equipment as is a stethoscope. My preference is for a distally lighted instrument 25 cm. long and ¾ to ⅞ of an inch in diameter. This, with a long (14-inch) grasper to hold cotton balls for sponging, is the only device needed in performing the examination. This procedure no longer is one to be done only by the specialist. It is not difficult nor very time consuming. The examiner need only to remember that the instrument

must be inserted slowly under direct vision and that force is never used. With these precautions, each individual has to teach himself its use.

The majority of patients relax well if they are told what to expect, and they should be told that they will note some abdominal distress as the instrument passes above the lumbosacral angle. In the occasional person, general anesthesia in the hospital may be necessary, but this is the exception rather than the rule. The knee-chest position is better in most cases than the inverted position requiring special tables. It allows the abdominal contents to fall toward the diaphragm without pressure against the belly wall and permits some straightening of the sigmoid flexures. It is true that occasionally a patient has died because the bowel was inadvertently perforated during the course of this examination, an incident for which I feel there is little excuse, but it has been said correctly that for every patient who has died because of sigmoidoscopy, a thousand have died because it was not done soon enough.

For patients presenting symptoms the same routine is carried out and in the order mentioned previously. Bleeding from the rectum may occur only occasionally, and hemorrhoids, fissure, or fistula may be present. All persons with vague but persistent abdominal distress that recurs should be studied, and since carcinoma of the right half of the colon (though prone to metastasize somewhat late) often produces blood depletion, every patient with an unexplained secondary hypochromic anemia should have this evaluation as well as a search for gastric carcinoma and diaphragmatic hernia. To the above routine is added, and as a final step in the order, a barium enema x-ray study. It is most difficult to convince the average physician that this should be the final step in patient evaluation rather than the first, but very often the radiologist gets this request prior to the insertion of an examining finger. Since definitive evaluation of the pelvic colon is usually impossible for the roentgenologist, owing to sigmoid flexibility and redundancy, he should refuse to examine the patient unless he has been presented with the sigmoidoscopic findings.

Steps in Examining the Patient with Symptoms.—

1. Careful, gentle, abdominal palpation
2. Digital examination
3. Determination of occult blood in the stool (guaiac test)
4. Proctoscopy
5. Sigmoidoscopy
6. Barium enema radiographic study
7. Double contrast enema study (in selected cases).

Roentgen examination of the colon is mandatory in every case where obvious reason for bleeding is found on rectal examination. Included are those cases with benign lesions such as hemorrhoids, even those manifest on digital or sigmoidoscopic examination; the likelihood of other lesions of the colon, in addition to those demonstrated in the pelvis, is quite great in these cases, and no efforts at therapy should be carried out until the status of the balance of the large bowel is known. Multiple primary tumors of the colon are not uncommon, and suspect areas should be defined and localized for the surgeon prior to any effort at excision. These facts are well known, but radiologists say that usually they are relied upon to make a diagnosis when no other method of investigation has been employed prior to their evaluation. The patient with a solitary small polyp within the range of the proctoscope or sigmoidoscope, even though it is asymptomatic, should have the benefit of this study. Multiple polyposis (usually a familial or hereditary condition) may thus be discovered in an asymptomatic person. This disease has almost a 100 per cent predilection for malignancy and demands drastic treatment when it is first discovered.

TREATMENT

For emphasis—all persons with benign anorectal lesions should have complete evaluation before any therapy is undertaken. So often after internal hemorrhoids have been eradicated surgically or by sclerosing therapy, continued bleeding at stool after complete healing has been found to be due to a malignant lesion higher in the bowel. The solitary small pedunculated polyp less than 1 cm. in diameter arising from the rectal or sigmoid wall should be removed through a scope in its entirety down to and including the mucosa underlying the pedicle, whether its surface is ulcerated or covered with apparently normal mucosa. Even if it is histologically malignant, provided there is no microscopic evidence of extension into the base of the pedicle, no other therapy may be necessary. Many cases of early malignant disease of this type may be cured by this simple procedure. However, record should be made of the location of the lesion, and recurrences should be searched for by endoscopic inspection of this and adjacent sites at 2-month intervals for at least a year and at increasing intervals thereafter. If histologic examination of the tissue removed shows extension into the mural base, more radical procedures are indicated as soon as possible, and they are managed in the same manner as is the mural ulcerative malignant neoplasm of the same location.

Obvious neoplasms within the range of the sigmoidoscope should have tissue removed for biopsy. Tissue should be taken from the edge of the mass rather than from its necrotic center. If possible, two or three small pieces should be secured for the pathologist for accurate diagnostic appraisal. Mural carcinoma at or below the lowermost portion of the peritoneal reflection in the pouch of Douglas, demands the radical surgical approach of an abdominoperineal proctosigmoidectomy as described by Miles in 1908. It was my privilege several years ago to hear a paper on this subject presented by the late Dr. Fred W. Rankin,[1] a leader in the world on diseases of the colon. This paper was discussed by Dr. Thomas E. Jones and by Drs. Leland S. McKittrick and Frank Lahey. Although several of these men had devised operative procedures for the management of this disease in this location, they had largely abandoned their own operations in favor of the Miles' procedure and they felt, as I do, that nothing has been devised since Miles described his operation that has its equal in successfully eradicating carcinoma of the rectum and rectosigmoid. This procedure entails a permanent abdominal colostomy, while several surgeons have devised sphincter-preserving pull-through operations to avoid colostomy existence. However, adequate parietal dissection deep within the pelvis usually destroys all or enough filaments of the nerves supplying the sphincter ani externus that it subsequently is paretic or paralyzed and the patient has thus been provided with a perineal colostomy which is far more difficult to manage. Where a successful end-result is obtained as far as control is concerned, adequate tissue adjacent to the tumor has rarely been excised, and recurrence of neoplasm is more common.

Here, a word must be said for colostomy. Properly constructed and properly placed, a colostomy is of little annoyance to the patient and requires but simple control. The surgeon should place it either through the main incision or through a separate stab incision so that it emerges through the abdominal wall about 1 inch below and 1 or 2 inches to the left of the umbilicus. If the initial stirrup is long enough (2 or 3 inches), and the aperture in the belly wall adequate about the bowel, there should be no retraction, prolapse, or stenosis. When these complications occur, operative revision of the stoma becomes necessary. A well-constructed colostomy functions nicely with dietary control and does not leak feces or intestinal gas. Colostomy bags and more complicated apparatus are not required; the patient wears a 4 inch by 4 inch gauze square on the center of which has been placed a little lubricating jelly to prevent its sticking to the mucosal rosette of the stoma. It is held in place with an encircling elastic band of home construction, and no bulky evidences of the colostomy's presence can be seen beneath the usual clothing. An adequate constipating diet, avoidance of gas-producing foods, such as beans, onions, melons, and members of the cabbage family, and alcoholic beverages, and reliance on creamed meats and vegetables and milk in large quantities, boiled if necessary, enables the remaining colon to be evacuated at regular intervals of 48 to 72 hours by gentle lavage with warm tap water enemas given through a rectal tube.

With adequate preoperative preparation of the patient in regard to water and protein balance, vitamin intake, blood transfusion, and the like, there should be a minimal mortality to the employment of the Miles' procedure in one stage. The reserve status of the patient at the conclusion of the abdominal stage should determine whether the perineal stage should be deferred for a few days.

The following is a method of bowel preparation that I have recently successfully used to eliminate accidental contamination of the peritoneal cavity with colonic content. About 24 hours before surgery, 2 ounces of castor oil are given on an empty stomach. One hour later and each hour for 4 doses 1 grain of neomycin is given. Two 500-mg. tablets of this antibiotic are then given by mouth every 4 hours for a total dosage of 10 grams. After initiation of this routine, nothing but water is given orally. This completely clears the colon of pathogenic organisms and no toxicity results, since the drug stays within the intestinal lumen and is not absorbed into the systematic circulation.

Metastatic spread of the disease to lymph nodes, liver, and tissues adjacent to the tumor cannot be determined with accuracy until exposure of the abdominal content at laparotomy. Liver metastasis was previously a contraindication to operation, but it is the consensus today that such involvement should not preclude surgical resection. Not every palpable mass within liver substance is a metastatic tumor. Solitary small lesions in the liver may be removed through a separate incision in the right upper quadrant. This may be done with a melon scoop; the cavity is packed with oxidized cellulose or absorbable gelatin sponge which is retained for hemostasis with mattress sutures of chromic catgut. Such a nodule may represent the only metastasis and this management may effect a cure.

Even in the presence of obvious multiple metastases, the patient subjected to a Miles' resection has a definite prolongation of life and often avoids the agonizing terminal discomfort that frequently ensues when the primary lesion is left in situ. Removal of the primary site in these cases usually slows the progression of the metastatic lesions, and there are patients who have survived 15 years after operative demonstration of extension to the liver.

Malignant lesions in the rectosigmoid and higher today are managed in most instances by anterior abdominal resection and primary end-to-end anastomosis. The studies of Gilchrist and David[2] have shown that distal retrograde lymphatic metastasis does not occur until all nodes proximal to the tumor have been replaced by anaplastic cells. The work of C. W. Mayo[3] has disclosed no evidence of distal spread in the bowel wall extending more than 2.5 cm. below the lowest palpable edge of the tumor. These findings have made anterior resection and anastomosis a safe curative procedure for utilization in many cases that would have otherwise necessitated a Miles' resection.

The first symptom of colonic tumor may be intestinal obstruction. Phenomena may vary from a complete inability to move the bowels with laxatives or enemas and absence of abdominal pain to acute manifestations of marked abdominal colicky pain and distention with nausea and vomiting. When this occurs, an exploratory film of the abdomen may differentiate by its gas and fecal pattern the type and location of the obstruction. For lesions that are thus considered colonic, a slowly given barium enema should localize the site of the tumor and permit a properly placed proximal decompressing colostomy, cecostomy, or ileostomy. This decompression of the proximal bowel and restoration of the patient to the status of a normal surgical risk must be done before any definitive treatment is given to the obstructing tumor. Exteriorization operations as suggested in the last century by Mikulicz are rarely indicated today, though, on occasion, there may be some utilization of this procedure for tumors of the splenic fixture. Decompression, sterilization of content, and primary resection with end-to-end anastomosis usually suffice. In these segmental resections, wedge removal of the associated mesocolon as far as the aorta is demanded in order to remove with the tumor all lymph nodes that might contain metastatic disease.

Tumors of the cecum and ascending colon are notorious for their lack of symptoms. Frequently symptoms develop only after the central necrosis has gone on to perforation and abscess formation, in which case they have been misdiagnosed as acute appendicitis and discovered with some astonishment at laparotomy. These neoplasms tend to be bulky, narrowing the lumen and exciting a protective adhesive wall outside the serosa. The first sign may be a painless mass in the lower right quadrant or right lateral abdomen with complete occlusion of the fecal stream (the content of the bowel here is liquid)—an abrupt mesenteric bowel ileus necessitating Miller-Abbott intubation and decompression with Wangensteen siphonage. Though these local neoplasms are relatively larger, in most instances, than tumors elsewhere in the colon, they metastasize late and, in spite of their bulk, are cured in most instances by ileocolectomy, removing the terminal 6 inches of ileum and the right half of the colon with wedge resection of the mesentery and node-bearing mesocolon. Intestinal continuity is restored by an end-to-end, end-to-side, or side-to-side ileocolostomy. Again, in almost every instance, this procedure

should be carried out in spite of visible and palpable extension of tumor to the liver.

The patient with multiple polyposis should be given the benefit of surgical care prior to the development of malignant degeneration. One with chronic ulcerative colitis has a predilection to develop polyps which often become anaplastic with exacerbations and remissions of this idiopathic process after years of conservative care. For both, the safest procedure is total colectomy in one or two stages, restoring the patient to health and safety with a permanent skin-grafted ileostomy.

CONCLUSIONS

1. Adequate, orderly investigation of the large bowel is necessary in all patients in order to arrive at early diagnosis of malignant lesions in the asymptomatic individual.

2. In the person with bleeding at defecation, with change in bowel habit, unexplained secondary anemia, or recurrent abdominal distress, diagnostic procedures should be utilized in a certain order prior to the institution of any therapy.

3. Sigmoidoscopy should always precede x-ray study of the colon, and this examination should be and is a part of the investigation carried out by the general practitioner.

4. A colostomy is compatible with a pleasant, happy, productive life.

5. There is a place for radical surgery of the large bowel in those individuals with liver metastasis.

6. Malignant lesions of the large bowel are very frequent and have a high rate of curability with existing surgical methods. This could be improved materially by adequate utilization of regular physical check-ups in the asymptomatic patient.

135 S. 17th St.

REFERENCES

1. Rankin, F. W., and Johnston, C. C.: Surgical treatment of cancer of rectum and rectosigmoid. J. Am. M. A. 136:371-375, Feb. 7, 1948.

2. Gilchrist, R. K., and David, V. C.: Consideration of factors influencing 5 year survival in radical resection of large bowel and rectum for carcinoma. Ann. Surg. 126:421-438, Oct. 1947.

3. Mayo, C. W.: Factors influencing selection of operation for carcinoma of lower portion of colon and rectum. Collect. Papers Mayo Clin. & Mayo Found. 40:167-171, 1948.

ADDITIONAL REFERENCES

Lahey Clinic, Boston: Surgical practice of Lahey Clinic. W. B. Saunders Co., Philadelphia, 1951.

Rankin, F. W., and Graham, A. S.: Cancer of colon and rectum. Ed. 2. Charles C Thomas, Springfield, Ill., 1950.

Anesthetic Evaluation and Management of the Poor-Risk Patient

FRANCIS J. SMITH, D.O., F.A.O.C.A.
Philadelphia

In dealing with the problem of anesthesia and care of the poor-risk patient it is impossible to speak in generalities because each patient must be treated as a specific problem, and plans for his care and administration must be individualized. The poor-risk patient is one who, after careful evaluation, shows marked deviation from normal in one or more of his several systems, the respiratory, cardiovascular, genitourinary, digestive, and nervous systems, or who has constitutional disease or disease of the blood or ductless glands.

The anesthetic agent and the method of administration should be so chosen that they will produce the least harmful effects on the altered physiology and at the same time prove adequate for the proposed operation.

In general, patients who are gravely ill are easy to anesthetize. The amount of anesthetic and the care with which it is given outweigh other considerations. What anesthetic to use and how to administer it will depend largely on the experience of the anesthetist. If the anesthetist's experience with various agents and technics is limited, he should select an agent and method with which he has had much experience. Up to the present there is little scientific evidence from the laboratory or from controlled clinical experiments to support the claim that any one general anesthetic is the best. This applies to the good-risk as well as to the poor-risk patient. A good airway, adequate oxygenation, and a level of anesthesia no deeper than that demanded by the operation are basic principles of safe anesthesia which are doubly important for the poor-risk patient.

By preceding the choice of a safe anesthetic procedure (there may be more than one) with careful preoperative preparation of the patient, and by carefully carrying out the procedure and accompanying it with suitable supportive measures during surgery and with proper postoperative care, it is possible to improve the status of the poor-risk patient from one of doubtful outcome to one which offers him more than a reasonable chance of satisfactory convalescence and recovery.

EVALUATION OF THE PATIENT

Too often much time is spent by the surgeon, internist, and anesthesiologist rehashing the irreversible processes that confront them. Such conditions as arteriosclerosis, hypertension, valvular and congenital heart disease, pulmonary fibrosis, emphysema, and liver and kidney disease fall into this category. At the same time reversible processes such as secondary anemia, hypoproteinemia, hypothrombinemia, dehydration, diabetes, and hypothyroidism are often neglected. A few basic facts which can be ascertained from a physical examination of the patient and the laboratory reports usually are all that is necessary to form a fair idea of the anesthetic and operative risk in a given case. Important among these are the blood count, the hemoglobin estimation, blood proteins, prothrombin time, the blood pressure, urinary findings, the degree of cardiac and renal insufficiency, and the general appearance of the patient in regard to nutrition, debility, and weight loss. A more exhaustive examination of the patient may show further evidence of physiologic abnormalities, but such information may not only be

unessential to the choice of the safest anesthetic agent but may even complicate the choice. There are some ordinary factors, however, which should be considered.

Age.—Anesthesia and operation are not tolerated as well by patients in the extremes of life as by those in the middle years. The respiratory system of infants and young children is easily depressed by anesthetic agents. Many of the aged are poor risks and do not react well to anesthesia. Debility and cardiovascular-renal disease are apt to be complicating factors in older people. The mortality rate of surgical patients who are more than 60 years of age is under some circumstances much greater than that of younger persons.[1] Elderly patients fare better after operation if they are gotten out of bed at the earliest possible moment.

Habits.—The mode of life may be a factor in the operative risk. Patients who use tobacco to excess and have accompanying bronchitis are prone to postoperative respiratory complications. Chronic alcoholics often react poorly under anesthesia and are more susceptible to postoperative complications. The same may be said of drug addicts, since in them nutritional and nervous disturbances are frequently encountered.

Nervous System.—Patients who are "high strung" usually are more difficult to anesthetize. Those who come to surgery worried and frightened do not present a good risk and frequently have a stormy convalescence. Adequate preoperative sedation and assurance are important therapeutic measures in these cases.

Debility.—Patients who are frail, debilitated, underweight, and poorly nourished lack the reserve of robust patients to withstand the toxicity of the anesthetic and the shock of the operation. Even small doses of the anesthetic depress the vital functions of these patients, and great care must be observed to prevent overdosage. Prolonged vomiting is serious since it leads to nutritional disturbances, decrease of glycogen in the liver, and the more serious condition of acidosis. The other extreme, alkalosis, is also serious and is sometimes produced by intestinal obstruction. The loss of gastric and intestinal secretion causes the blood chloride and sodium figures to fall and the nonprotein nitrogen elements to rise, and alkalosis may develop.

Hepatic Function.—The liver and its function have an important bearing on the choice of anesthesia. One of its principal functions is its power of detoxification, including detoxification of certain anesthetic agents. Liver function is inhibited and liver tissue damaged by certain anesthetic agents. The normal functioning liver, however, stands the strain imposed upon it by the anesthetic and the operation remarkably well if its store of glycogen is adequate and anoxia is prevented. If its function has been impaired by toxemia and if nutritional disturbances have depleted its supply of glycogen, it not only performs its function poorly but is vulnerable to further damage from the anesthetic. The most toxic anesthetic agent to the liver is chloroform. Vinethene may also damage hepatic tissue, especially if administration is prolonged. Ether impairs the function of the normal liver only slightly, and normal function is soon restored during the postoperative period. Cyclopropane and the short acting barbiturates have little if any harmful effect on liver function in the presence of adequate oxygen. Nitrous oxide as the sole or principal agent is contraindicated in impaired liver function. The danger lies in the anoxia necessary to secure surgical anesthesia. Clinical evidence has not proved that Pentothal sodium causes any appreciable damage to the liver or increases damage already present. Patients with severe liver disease should be prepared for operation by the administration of a high caloric diet, high in carbohydrates, low in fat.

Renal Function.—Ether anesthesia causes a marked decrease in urinary output. Normal function is restored, however, during the first 24 hours after operation. Cyclopropane also suppresses urinary output during anesthesia, but this is followed by a compensatory increase in excretion following operation. If renal damage is present at the time of operation, anesthetic agents which are toxic to renal tissue and tend to inhibit renal function will produce more serious disturbances in those organs. The offending agents are chloroform, ether, Vinethene, Avertin, and Pentothal, in the order given.

Cardiovascular System.—Cardiac insufficiency if advanced and uncompensated presents one of the most serious complications the anesthetist is called upon to face. Cardiologists agree, however, that patients with many types of cardiovascular lesions stand anesthesia unusually well, provided there is adequate compensation and cardiac reserve is satisfactory. Although a careful cardiovascular examination should be routine before operation, especially in poor-risk cases, a fair idea of the patient's cardiac reserve may be obtained by applying certain critical tests.

Experience has shown that Moots' index or formula provides an approximate index to the cardiac reserve. It is obtained by multiplying the pulse pressure by 100 and dividing by the diastolic pressure. The result is 50 or thereabout in the normal subject. For example: If the blood pressure is 120/80 and the pulse pressure is 40, multiply 40 by 100 and divide by 80, the diastolic pressure, and the answer obtained is 50, or 50 per cent. Patients showing an index between 40 and 60 per cent apparently have ample cardiac reserve. If the index is between 25 and 40 or between 60 and 75, the amount of cardiac reserve is low. If the index is below 25 or above 75 the case is probably inoperable, the cardiac reserve being too low.

The simplest and probably the most reliable of these tests is the breath-holding test of Henderson. This is especially emphasized because it may not only indicate the degree of cardiac reserve but may also disclose such conditions as acidosis and respiratory or blood diseases. The test is performed after the patient has been lying down for 15 minutes or more. After observing several normal respirations, at the end of a normal expiration the physician should pinch the patient's nose tightly and instruct him to keep his mouth closed and to hold his breath as long as possible. Normally one should be able to hold the breath for 25 to 30 seconds. Severe acidosis may be expected when breath-holding lasts only 5 to 10 seconds. Oxygen starvation which initiates even the slightest anoxemia or acidemia is dangerous in patients suffering from hemoglobinemia, shock, cardiorenal or pulmonary lesions, or goiter or other disturbances of metabolism in which there is a tendency to acidemia.

These two tests which are almost unknown or forgotten in some quarters are just as valuable today as they were 25 years ago. Of course it must be realized that anesthetic tolerance must not be judged upon one isolated index or formula, but should be determined from the maximum of information gained by the anesthetist. When the aforementioned tests are not made, it may be said with less accuracy that if there is no cardiac pain or dyspnea, either with or without moderate exertion, and no pretibial edema or rales in the

bases of the lungs, the heart will probably stand the strain of an operation. The risk increases in proportion to the extent the patient's activities are curtailed by the cardiac condition. A survey of the literature indicates that it is generally agreed that angina pectoris, coronary occlusion, congestive failure, and syphilitic aortitis cause high operative mortality. Also, it is agreed that valvular heart disease and auricular fibrillation, if unaccompanied by failure and tachycardia, do not greatly increase the anesthetic or the operative risk. Decompensated cardiacs must be treated with bed rest, digitalization, and restriction of sodium in preparation for surgery.

Respiratory System.—Diseases of the lungs or of the respiratory passages have a very important bearing on the course of anesthesia, the complications that may arise, and the degree of risk for the patient, both during and after the operation. There is also the added risk of producing other pulmonary complications as a result of the anesthesia and surgical procedure or of producing an extension of those conditions which existed prior to surgery. The closer the vital capacity approximates the tidal air, the graver the risk. Vital capacity has been defined as the quantity of air that can be expelled after the greatest possible inspiration, normally about 2,700 cc.; tidal air is the quantity of air taken in and expelled by an ordinary respiration, normally about 500 cc. Lesions which affect vital capacity are emphysema, abscess of the lung, tuberculosis, pneumonia, pleurisy with effusion, asthma, and certain cardiac conditions, especially mitral stenosis. A reduction in vital capacity always calls for particular care in maintaining adequate ventilation during anesthesia. Where there is any question of furnishing an adequate supply of oxygen, an endotracheal tube with inflatable cuff should be introduced under direct vision. Five to 8 minutes of severe anoxia will produce severe and irreparable damage to the cerebral cortex.

Suction aspiration of the tracheobronchial tree when indicated during the immediate postoperative period is assuming major importance in the control and prevention of postoperative pulmonary complications. Prominent among these complications are bronchopneumonia, atelectasis, and massive collapse of the lung. The administration of oxygen has become routine treatment postoperatively, especially in poor-risk cases, and is given by tent, nasal catheter, or special mask.

Diabetes.—The diabetic, for the most part, has been taken out of the group of poor-risk patients by modern diabetic therapy and improved anesthesia. Unless these patients have another disease which in itself would increase the danger of anesthesia and of operation, their outlook is generally favorable. Other conditions do frequently exist in diabetics, among which is the tendency to coronary thrombosis and cerebrovascular accidents. Many clinicians are of the opinion that the controlled diabetic can even withstand the effects of such agents as ether, but I personally am of the opinion that one is justified in using only those agents which interfere least with carbohydrate metabolism. Ether not only increases the blood sugar level but also has a tendency to produce or aggravate acidosis as a result of postoperative vomiting and the consequent restriction of food. Nitrous oxide and cyclopropane are suitable because they affect carbohydrate metabolism but slightly. Spinal and regional anesthesia are also satisfactory. Pentothal sodium alone or in combination with local, regional, or spinal anesthesia or in combination with inhalation anesthesia may also be indicated.

SELECTION OF AN ANESTHETIC AGENT

There is a difference of opinion among anesthesiologists as to the best anesthetic agent and method for the poor-risk patient, just as there is a difference of opinion among surgeons as to the best surgical procedure for the correction of surgical ailments of these patients. In evaluating the available anesthetic agents it is quite obvious that the ideal anesthetic has not yet been found.

Ether.—Valuable as ether is in anesthetic practice, its use should be avoided as much as possible in poor-risk cases, owing to the irritating effects on the respiratory tract which predispose to postoperative pulmonary complications and because of the vomiting, dehydration, acidosis, and hepatic and renal dysfunction it causes. On the other hand, more oxygen may be given with ether than with any other inhalation agent. If it is necessary for an inexperienced person to administer an inhalant anesthetic, ether is the safest. Ether, too, will often eliminate the arrhythmias of cyclopropane.

Cyclopropane.—The value of cyclopropane in many types of poor surgical risk is incontestable on the basis of its low toxic effect on liver and kidneys, its anesthetic potency, and the absence of irritation to the respiratory system. It is particularly indicated in thoracic surgery, in thyroidectomy, and in severely shocked and toxic patients. It is also indicated in chronic bronchitis, asthma, and respiratory obstruction. In asthma and respiratory obstruction, 20 per cent helium can be added to advantage. Because the gas does not produce liver damage or kidney disease, it is useful in eclampsia. Cyclopropane can also be used as an adjuvant to nitrous oxide-oxygen anesthesia if more relaxation is required. Cyclopropane is quite explosive as well as inflammable and is seldom used in the hospitals of the Philadelphia College of Osteopathy because of the difficulty in securing the cooperation of all those persons necessary to make its use entirely safe.

Nitrous Oxide.—On the basis of its low toxicity nitrous oxide-oxygen is safe, but the anoxemia which may occur has toxic effects. The gas is not suitable for major procedures since the concentrations which are necessary are high enough to cause asphyxia. Asphyxia as a method of anesthesia is condemned since it causes respiratory and cardiac depression, elevation of the blood pressure, and, occasionally, acidosis. When used with at least 25 per cent oxygen in cardiac patients, nitrous oxide produces a rapid and smooth induction. It should be avoided as a primary anesthetic in cardiac disease but may be combined with ether, cyclopropane, basal narcosis, intravenous barbiturates, or a muscle relaxant for conduction, provided there is no hypoxia. In advanced heart disease in which hypoxia is already present, even slight reduction in oxygen concentration may cause serious anoxia with resulting coronary or cerebral insufficiency.

The Muscle Relaxants.—The use of muscle relaxants has produced a marked change in clinical anesthesia. Agents are now available whose almost exclusive function is to provide muscular relaxation. These muscular relaxants produce relatively few undesirable side-effects. Their greatest value lies in the fact that they permit the use of lower concentrations of the more potent and toxic agents and permit wider employment of the less potent agents with lighter planes and anesthesia, resulting in greater margins of safety for the patient. The dangers attending the use of relaxants are

chiefly those associated with decreased pulmonary ventilation, a condition which the anesthesiologist can easily correct during clinical anesthesia.

The employment of curare as a safe and useful drug was made possible by Gill[2] who brought it out of the jungles of South America and by Griffith and Johnson who introduced it into the clinical practice of anesthesiology.

The principal muscle relaxants are d-tubocurarine chloride (obtained from crude curare in 1943), Flaxedil, Syncurine, and succinylcholine chloride. All of them produce muscle relaxation by action at the neuromuscular junction. The first two interfere with depolarization of the postjunctional membrane by acetylcholine, and are antagonized by neostigmine and Tensilon. The latter two, on the other hand, effect a depolarization of the postjunctional membrane, and are not antagonized by neostigmine or Tensilon. Of the four relaxants mentioned, I shall briefly discuss d-tubocurarine and succinylcholine.

The muscle relaxants do not produce unconsciousness except when an overdose has been given, nor do they possess any analgesic property; hence when unconsciousness is desired or relief from pain is necessary, it must be obtained with an appropriate anesthetic agent and method. D-tubocurarine may be used with any type of anesthesia: general, local, or spinal. The dose ranges from 1.5 cc. to 5 cc. (4.5 mg. to 15 mg.) given intravenously, and the effect lasts from 20 to 40 minutes. Succinylcholine exhibits outstanding flexibility and low toxicity. It provides the greatest degree of controllability among the muscle relaxants and is the ultrashort-acting agent of the group. The intensity of effect can therefore be varied rapidly by varying the rate of administration of the agent in an intravenous drip, which is the preferred method of employing it. Succinylcholine is characterized by a lack of undesirable side-effects referable to any vital system, and there is no cumulative effect. There is no effective antagonist. Succinylcholine requires closer attention during administration than other agents but provides greater controllability. It most nearly approaches the ideal muscle relaxant. It is the agent of choice, especially in the aged and debilitated patient, and whenever rapid loss of its effect is important.

Pentothal Sodium.—This drug may be used in short minor surgical procedures and for longer operations not requiring too much relaxation, but it should always be supplemented with sufficient oxygen. Intravenous Pentothal is frequently used to produce a satisfactory induction to inhalation anesthesia. It also can be combined with a muscle relaxant, and intercostal block or spinal anesthesia. These combinations of agents and methods reduce the toxicity of the drug and give a high degree of oxygenation. Lundy[4] was the first to advocate this balanced or combined anesthesia in impaired risk patients and demonstrated that this method of anesthesia is satisfactory in a wide range of cases. A slow careful induction with nitrous oxide and oxygen followed by intravenous Pentothal provides a satisfactory combination, if relaxation is adequate. Equal parts of nitrous oxide and oxygen increase the percentage of oxygen in the inspired air and materially decrease the amount of Pentothal required. Excessive preanesthetic sedation should be avoided, especially in cardiacs. The barbiturates and opiates lower the minute volume of respiration so that the alveolar air is renewed infrequently, and severe anoxia may develop. Because of increased usefulness and safety, the trend is toward employing intravenous anesthesia in combination with other agents and technics for major surgery rather than as the sole or principal agent.

Spinal Anesthesia.—There is a wide difference of opinion concerning the use of spinal anesthesia in poor-risk cases. Spinal anesthesia may be indicated in many so-called poor-risk cases but contraindicated in others. Indications and contraindications in anesthesia are relative, rarely absolute. Disease of the central nervous system is usually considered a contraindication to spinal anesthesia, the assumption being that spinal anesthesia may diminish resistance to the disease, but more frequently it is considered a contraindication because the patient may attribute any new symptoms to the anesthesia. Increased experience in the use of the newer technics in spinal anesthesia, which give better control of the height and duration of anesthesia, make it possible to use spinal anesthesia in many cases where it formerly was considered to be contraindicated. By careful watching of the patient and by immediate institution of measures to support depressed respiratory and circulatory mechanisms, many patients to whom spinal anesthesia was formerly denied because the risk was considered too great may now be successfully managed.

The presence of severe hemorrhage, anemia, shock, cardiac decompensation, or coronary disease was formerly considered a distinct contraindication to spinal anesthesia. With the use of fluids preoperatively and transfusions in the case of severe hemorrhage, anemia, and shock, and adequate preoperative medical control of cardiac decompensation and coronary disease, combined with proper supportive measures during anesthesia as previously mentioned, the dangers presented by these cases are considerably lessened.

Furthermore, there are certain conditions, notably intestinal obstruction with shock, in which the improved operating conditions afforded by spinal anesthesia overbalance any increased hazard which it might present. Also, spinal anesthesia may prevent early shock thought to be caused by reflex action.

Spinal anesthesia is particularly indicated in the presence of disease of the liver, the kidneys, and the respiratory tract. Similarly, metabolic diseases such as diabetes constitute an indication for spinal anesthesia because it produces little, if any, disturbance of the blood sugar level, of reaction of body fluids, and of other metabolic processes. Spinal anesthesia also finds a field of usefulness in poor-risk cases in operations in the abdomen and on the lower extremities.

These poor-risk patients must be well oxygenated at all times, and they require much less of any anesthetic agent to produce comparable anesthesia than does a vigorous husky youth.

CONCLUSION

Although great progress has been made in providing more adequate and safer anesthesia for the poor-risk patient, much remains to be accomplished if the morbidity and mortality of this class of patient is to be lowered still further. It is important to stress that the patient's pathologic physiology should be considered at all times, that is, in the preoperative stage, during the operation and anesthesia, and in the postoperative stage. Close attention to this will result in a lessened mortality following operation and also in a smoother postoperative course and lessened morbidity. The anesthetist can contribute to the improvement by being ever careful of his choice and application of the available anesthetic agents and methods. However, the safe-

ty and well-being of the patient depend, not upon the anesthetic agent, but upon the anesthetist. Only by close cooperation and active consultation between the surgeon, the internist, and the anesthesiologist can the operative and postoperative complications that are so apt to occur in these patients be prevented.

The Vernon House
6445 Greene St.

REFERENCES

1. Carp, L.: Basic principles in geriatric surgery. Ann. Surg. 123: 1101-1110, June 1946.
2. Gill, R. C.: Curare: misconceptions regarding discovery and development of present form of drug. Anesthesiology 7:14-23, Jan. 1946.
3. Griffith, H. R., and Johnson, G. E.: Use of curare in general anesthesia. Anesthesiology 3:418-420, July 1942.
4. Lundy, J. S.: Clinical anesthesia. W. B. Saunders Co., Philadelphia, 1942.

ADDITIONAL REFERENCES

Chivers, E. M., and Evans, W. E. F.: Anaesthesia in surgical shock. Brit. J. Anaesth. 17:92-97, Jan. 1941.
Cullen, S. C.: Clinical and laboratory observations on use of curare during inhalation anesthesia. Anesthesiology 5:166-173, March 1944.
Griffith, H. R.: Physiological and clinical action of curare. Anesth. & Analg. 25:45-51, March-April 1946.
Gross, H., Stein, P., and Buchberg, A. S.: Surgical anesthesia, heart disease and cardiovascular responses. Anesth. & Analg. 30:250-261, Sept.-Oct. 1951.
Haugen, F. P.: Hypertension; management of anesthesia period. Anesth. & Analg. 22:152-157, May-June 1943.
Lorhan, P. H.: Anesthesia and surgery in patients of advanced age. Anesth. & Analg. 28:190-202, July-Aug. 1949.
Morgan, A. D.: Shock and anesthesia in relation thereto. Anesth. & Analg. 22:81-92, March-April 1943.
Nicholson, M. J.: Anesthesia for poor risk patient. Anesth. & Analg. 28:98-104, March-April 1949.
Poindexter, C. A.: Evaluation of cardiac status of surgical patient. M. Clin. North America 22:1505-1511, Sept. 1938.
Saklad, M.: Grading of patients for surgical procedures. Anesthesiology 2:281-284, May 1941.
Schuhmacher, L. F., Jr., and Eversole, U. H.: Technics of spinal anesthesia. Anesthesiology 3:630-643, Nov. 1942.
Whitacre, R. J., and Fisher, A. J.: Clinical observations on use of curare in anesthesia. Anesthesiology 6:124-130, March 1945.

Nutritional and Electrolyte Preparation of the Poor-Risk Surgical Patient

MORTON TERRY, B.A., D.O., M.Sc.
Miami

During recent years the physician has been introduced to a truly scientific approach to the problem of the maintenance of proper nutritional and electrolyte status of the sick. The advent of these clinical and biochemical advances has been reflected in the increasing survival rate of critically ill patients and those subjected to extensive surgical procedures. It has been speculated that more lives will be saved by proper use of water and electrolytes than by most other therapeutic measures.[1-4]

The purpose of this paper is to discuss the methods of preparing for surgery the patient who is considered a poor risk from the nutritional standpoint. Specific nutritional deficiencies resulting from metabolic diseases such as hyperthyroidism and diabetes mellitus will be omitted.

WATER

The simplest but most important of all food elements is water which is, of course, indispensable to life.[4,5] The dehydrated patient is always a poor surgical risk.[6] The sunken face and its expression; the dry, hot, and turgid skin; dryness of the buccal mucosa and the tongue; thirst; and possible elevation of temperature are familiar signposts. The recent history is important, and the laboratory may help by demonstrating an increase in any of the following averages:

Hematocrit: males 42 to 50 per cent; females 39 to 43 per cent
Blood specific gravity: males 1.0566; females 1.0533; daily range 0.0033
Plasma specific gravity: 1.0270; daily range 0.033
Plasma protein: 7 grams per 100 cc.; range 5.9 to 7.9 grams per 100 cc.[7]

Although recent studies have revealed newer values,[8-13] Table I demonstrates simply the approximate distribution of the body water and Table II the normal water loss.

The three fluid compartments are in constant equilibrium with one another, and, in an attempt to maintain osmotic normality, adjustments are rapidly made through the selectively permeable membranes which separate them. It is obvious that water needs will be increased by any circumstances which lead to greater insensible losses and which increase the need for a larger volume of urine (febrile states, environmental temperatures, et cetera). To this can be added water loss from other sources such as vomiting, diarrhea, fistula drainage, exudates, and paracentesis withdrawal. It then becomes a relatively simple procedure to determine the amount of water replacement necessary each day by all routes. A careful intake and output record by the nursing personnel is mandatory to achieve proper hydration. The clinical findings and laboratory procedures listed above as well as observation of the urinary specific gravity will inform the internist or surgeon when the dehydrated patient is ready for surgery.

TABLE I

Body Weight 70 Kg. (154 lbs.)		
Total water 70%	=49,000 c.c.	
Intracellular fluid 50%	=35,000 c.c.	ICF
Extracellular fluid 20%	=14,000 c.c.	ECF
ECF-interstitial fluid 15%	=10,500 c.c.	ISF
plus intravascular fluid 5%	= 3,500 c.c.	IVF

TABLE II

Approximation of Daily Obligatory or Physiologic Water Loss	
	c.c.
Urine	1,000
Insensible (skin and lungs)	1,500
Stool	100
Total	2,600

ELECTROLYTES

Replacement and maintenance of the electrolyte ions in the various body fluid compartments is not as easily achieved as there is no simple formula to follow

nor is there any constant daily requirement which can be filled.[14,16-22] Table III demonstrates the distribution of electrolytes in the intracellular and extracellular body fluid.

TABLE III
Normal Body Electrolyte Concentrations

Extracellular mEq./L. Plasma						Intracellular mEq./L intracellular water			
Cations			Anions			Cations		Anions	
Na	142	(0.435)	Cl	140	(0.282)	K	150	HCO_3	10
K	4.5	(0.256)	HGO_3	29	(0.466)	Na	10	Cl	2
Ca	5	(0.500)	SO_4	1.5	(0.625)	Mg	40	HPO_4	88
Mg	3	(0.833)	HPO_4	2	(0.580)			Protein	80
			Org. acids	2				SO_4	20
			Protein	16	(2.43)				

The term "milliequivalents per liter" is employed rather than "milligrams per cent" as only the former indicates a relationship between the positively and negatively charged ions. To avoid reference to tables for atomic weights and extra mathematics, a conversion factor is listed with each electrolyte. To obtain milliequivalents per liter one need only multiply the electrolyte concentration in milligrams per cent by the appropriate conversion factor.[23]

Time does not permit a detailed discussion of the regulatory mechanisms within the body for water, electrolytes, and acid-base balance and the effects the endocrine system may have on them. Some of the more common states of electrolyte imbalance will be briefly discussed:

Dehydration.—

a. Hypotonic. Electrolyte loss is greater proportionately than the fluid loss. This condition occurs when large quantities of fluid are administered with little or no electrolytes. The concentration of serum sodium and chloride diminishes and the urine becomes dilute. There is weakness, pallor, decreased cardiac output, and lowered blood pressure but no thirst.[24] The treatment is, of course, administration of saline to replace the sodium and chloride and water which have been lost from the extracellular compartments.

b. Hypertonic. Water loss from the extracellular compartments is great and thus the electrolytes are concentrated. Excessive perspiration, high fever, and renal conditions with polyuria without fluid replacement are usually causative. Although there is generally no alteration in the pulse or blood pressure there is scanty, concentrated urine, thirst, dry skin and tongue, and fever. Glucose or protein in water is the obvious treatment in these cases.

Hyponatremia.—Deficiency of sodium intake, marked perspiration, renal disease, Addison's disease, and excessive loss from the gastrointestinal tract may be the causes.[25] Dehydration, low or absent urinary chlorides, elevation of urea nitrogen due to reduced glomerular filtration, vertigo, nausea, and, finally, stupor and convulsions may be seen.[26]

Hypernatremia.—Administration of an overabundance of salt is the common cause,[22] but desoxycorticosterone, ACTH, and cortisone may be responsible as can congestive heart failure.[25] Tissue edema is the common manifestation. Although plasma sodium levels over 155 mEq. per liter are diagnostic, occasionally the condition can exist with normal concentrations.

Hypochloremia.—Chloride is most commonly lost from the body as a potassium salt;[1] thus its deficiency generally is associated with hypopotassemia and hyponatremia and therefore the symptoms would be those of the primary conditions. In hyponatremia, altered renal function is the cause. Chloride deficiency may occur independently in those cases where there is excessive gastric hydrochloric acid loss as from emesis, suction, lavage, or fistula. This circumstance would result in alkalosis with an elevated carbon dioxide combining power and a serum chloride level of below 90 mEq. per liter. Treatment consists of administration of sodium, potassium, or ammonium chloride or a combination of these solutions.

Hypokalemia.—

Potassium is lost in alkalosis,[27] cellular destruction,[28] inadequate intake prior to and following surgery, stress, vomiting, diarrhea, intestinal and gastric suction, and fistula. Hypopotassemia increases gastric secretions, thus further increasing the electrolyte loss.[29] Approximately 2.4 mEq. of potassium are lost for every gram of nitrogen lost from tissue breakdown as from starvation. Possibly the greatest danger is that of administration of large amounts of potassium-free glucose and saline, which forces sodium into the cells and potassium into the extracellular compartment where it is excreted. Diabetic acidosis, ACTH and cortisone therapy, and familial periodic paralysis are other causes of potassium depletion. The clinical picture which is now well known and appears when the serum potassium level drops below 3 mEq. per liter includes generalized muscular weakness, listlessness, abdominal distention, flaccid paralysis, muscular twitching, and electrocardiographic changes (depression of ST interval, low amplitude and inversion of T waves, prolongation of QT interval, and prominent U waves).[1,2,28]

In treatment, care must be taken to correct the associated electrolyte losses concomitantly with the administration of potassium. A safe method is that of including 40 mEq. per liter of potassium in solutions until the patient's serum potassium has been normal for 24 hours; of even greater benefit would be the prophylactic administration of potassium where loss is anticipated and replacement as it is lost.[30] It must be remembered that sudden rises in serum potassium levels are dangerous and thus potassium must be administered with care. Glucose administered simultaneously accelerates the passage of potassium to the intracellular compartment.

Hyperkalemia.—Increased serum potassium (over 7 mEq. per liter) occurs from excessive administration, particularly in cases in which urinary output is diminished. Excessive tissue breakdown may also be responsible. Anuria, paresthesia of the hands and feet, weakness, and electrocardiographic changes (elevation of T with narrowing of the base, widened QRS, and disappearance of P wave) may be noted.[31] Treatment consists of administration of large amounts of glucose, insulin, and cation exchange resins.

Acidosis.—Metabolic acidosis is due generally to depletion of body cation (sodium, potassium, and calcium) which most commonly is due to fluid loss from the lower intestinal tract. The extracellular fluid space shrinks, causing diminished renal function which causes increased retention of acid ions (sulfate and phosphate). Ketone bodies displace bicarbonate from the plasma, and the carbon dioxide combining power drops to 18 mEq. per liter or below. Treatment consists of administration of sodium, potassium, and chloride along with sodium lactate. Respiratory acidosis can occur with any mechanism which causes retention of carbon dioxide.

Alkalosis.—

Metabolic alkalosis may result from excessive ingestion of sodium bicarbonate or an excessive loss of chloride ion. Potassium shifts from the intracellular to the extracellular compartment and is lost in the alkaline urine. Excessive loss of gastric juice is a common cause, and thus it might be suspected after prolonged vomiting as from duodenal obstruction. The carbon dioxide combining power is more than 32 mEq. per liter. Treatment consists of the administration of sodium and potassium chlorides.

Magnesium, calcium, and phosphate deficiencies may also occur but are not so common, particularly since many of the manufacturers of repair solutions now include these elements in their products.

Tables IV, V, VI, and VII contain general information which may be of value in calculating patient needs.

TABLE IV*

Normal Serum Electrolyte Levels

Calcium	4.5-5.5 mEq./liter
Chloride	100-110 mEq./liter
Magnesium	1.5-3.0 mEq./liter
Phosphorus	
Children	2.3-3.8 mEq./liter
Adults	1.8-2.3 mEq./liter
Potassium	3.5-5.5 mEq./liter
Sodium	137-147 mEq./liter

TABLE V*

Normal Adult Secretions Over 24 Hr. Period

Saliva	1,500 ml.
Gastric secretion	2,500 ml.
Bile	500 ml.
Pancreatic juice	700 ml.
Intestinal mucosa	3,000 ml.

TABLE VI

Electrolyte Loss (mEq./liter) from Common Sites

	Na	K	Cl	HCO_3
Gastric asp.	35	12	150	0
Intestinal fluid	140	10	105	25
Bile	140	10	100	30
Pancreatic juice	140	10	75	75

TABLE VII

Aid in Calculating Electrolyte Values

1 gm. NaCl = 17 mEq. of Na and Cl
1 gm. KCL = 13 mEq. of K and Cl
1 gm. Na = 43 mEq.
1 gm. K = 26 mEq.
1 gm. Cl = 29 mEq.
500 cc. blood contain 40 mEq. Na, 16 mEq. Cl, 2 mEq. K.

VITAMINS

Although vitamin deficiencies offer no therapeutic problem owing to ease of administration, availability of fine products, and lack of toxicity from overadministration, the existence of deficiency states in debilitated and poor-risk patients must be kept in mind. Vitamins are not food substances but catalysts in the process of food utilization and thus must be employed in conjunction with other nutrients.[5] The history will reveal much of the dietary background, and the physical examination and laboratory tests will furnish the remainder of the information in most vitamin deficiencies and in those cases in which alcoholic hepatic cirrhosis and other circumstances may require excessive vitamin supply.

Supplementary vitamin C should be administered orally or parenterally in all surgical cases as its importance in wound healing has been definitely established.[32] Recent investigations of the adrenal gland and the alarm reaction due to depletion of ascorbic acid from the cortex make the need even more urgent. The normal surgical patient should receive 200 mg. daily, but the malnourished individual requires 1 gram per day, and in cases of questionable intestinal function administration should be parenteral.

Intravenous glucose, particularly in the presence of vomiting or diarrhea and fever, rapidly causes thiamine deficiency in malnourished or dehydrated patients. As thiamine is required for glucose metabolism, the body store is quickly depleted by the sudden introduction of large amounts of glucose. This vitamin has also been demonstrated to play a part in wound healing, and evidence is present that it increases the resistance to experimental shock from hemorrhage. Overdosage is apparently harmless and would be advisable in the poor-risk patient.

The role of vitamin K is so well known as is its relation to biliary and hepatic disease that no detailed discussion is needed. Riboflavin is important in carbohydrate and protein metabolism, and a minimum of 2 mg. daily should be supplied parenterally. Approximately 50 mg. of niacin should be given daily. Vitamin A administration has resulted in acceleration of wound healing after experimental administration.

ENERGY (Caloric) NEEDS

In supplying caloric needs, carbohydrate has the advantage of being completely combustible and leaving no nitrogenous residue which will be a burden on the kidney. It also supplies water as a by-product of its utilization. Approximately 25 calories per kilogram are required for the average patient at bed rest.[33] This may be increased to 35 to 40 calories per kilogram in depleted individuals, and an increase of 7 per cent is necessary for every degree of fever. In the patient who can eat, fat supplies twice as many calories per gram as does glucose and adds palatableness to the diet, Glycosuria will not occur with intravenous dextrose administration if its rate does not exceed 0.6 gram per kilogram of body weight per hour. Fructose has been widely used of late and has the advantage that larger concentrations can be infused more rapidly without the occurrence of glycosuria and the attendant diuresis.[34,35] It is claimed that more rapid and efficient liver glycogen formation,[36,37] conversion to glycogen without insulin,[38] and better utilization in fasting or starvation states[39] occur with fructose than with dextrose.

The use of alcohol in parenteral fluid therapy has been increasing in popularity. A 5 per cent alcohol, 5 per cent glucose solution will yield 480 calories per liter in contrast to an equal amount of 10 per cent glucose or fructose which supplies 400 calories. Alcohol is protein sparing,[40] and the daily metabolism is 3 to 4.5 grams per kilogram of body weight.[41] One cc. of absolute ethyl alcohol will yield 5.6 calories.

Parenteral injections of fat emulsions are being administered experimentally,[42] and oral preparations have been used quite successfully.[43]

PROTEIN

Protein is the most recent addition to the solutions used in parenteral alimentation,[4] and its metabolism is quite complicated.[44] Elman[5] feels that it is the most common of all nutritional deficits in the surgical patient. In the patient who is unable to eat, carbohydrates will spare only a portion of body protein breakdown.

*Reprinted by permission from "Parenteral Fluid Therapy," Baxter Laboratories, Morton Grove, Ill., 1953.

Several important basic observations have been made. Incomplete mixtures of amino acids cannot be utilized in the synthesis of body protein even when the missing essential amino acid is provided 6 hours later.[45,46] For cell anabolism the essential amino acids must be available simultaneously, and amino acids are not stored in the body for much longer than an hour.[45,47]

The scope of this paper does not permit discussion of the altered physiology in the protein-deficient patient or the details of regeneration or the important role protein plays. Laboratory measurements of the serum or plasma protein levels in themselves are frequently very unsatisfactory as it is not uncommon for a severely deficient individual to have normal levels because of the decrease in total blood volume.

Basically, protein deficiency must be considered under three divisions: hemoglobin, plasma protein, and tissue protein. The most efficient, rapid, and simple correction of the first type is whole blood transfusion, and this is so simply performed that no further discussion is needed. The hemoglobin deficiency should be corrected first and improvements in the blood count and the hematocrit value are generally sufficient criteria of its correction. Plasma infusions and human albumin will help materially in correction of the plasma protein deficiencies although only a relatively small portion remains in the blood stream. The third concern is the provision of adequate amino acid mixtures to correct tissue protein deficit. It is important to note that plasma infusions are a wasteful, roundabout method for this purpose as the plasma proteins must first be hydrolyzed to amino acids and then resynthesized to tissue protein. A minimum of 100 grams of protein per day should be supplied. This amount will not correct deficiencies but will maintain the daily needs and prevent existing states from becoming more severe. Appropriate amounts of carbohydrate, vitamins, fluids, and minerals must also be administered.

Many satisfactory amino acid solutions are available for intravenous administration. It has been previously stated that amino acids are not stored and that synthesis of protein molecules is not accomplished unless all the necessary elements are present simultaneously. Intermittent feeding, orally or intravenously, will not supply the excessive demands for protein in the sick patient.[48] For these reasons for the past 6 months I have frequently used continuous enteral tube feeding and results have been very satisfactory. The advent of the narrow gauge polyvinyl tubing has eliminated most of the discomfort and technical difficulty of passing the tube or allowing it to remain in place for long periods. Two commercial preparations, Sustagen, a powder which is dissolved for use, and Dripolac, a stable sterile solution, have been used. Both supply a well-balanced preparation of carbohydrate and protein, but I prefer the second because of its ease of administration. It has the added advantage of being supplied in a regular and a low sodium type but it must be supplemented with vitamins. Each liter supplies 900 calories, and this total can be increased by adding ethyl alcohol, 65 grams of protein, and well-balanced electrolytes. The patient may eat with the tube in place and from 1 to 3 liters per day are administered according to need, the rate being accurately controlled with clamps and visual drip chamber. The only adverse reaction I have observed has been diarrhea which generally could be managed by altering the rate of administration and by the use of antidiarrheal agents. It is my opinion that this is the method of choice in many instances in which the intestinal tract is available for feeding and that all the elements necessary for proper nutritional and electrolyte preparation and maintenance of the poor-risk surgical patient can be supplied without using the intravenous route.

SUMMARY

1. Fluid, electrolyte, vitamin, carbohydrate, and protein needs of the adult surgical patient have been outlined.

2. Methods for the correction of these nutritional maintenance deficiencies have been discussed.

3. Special mention has been made of the favorable results obtained with the use of continuous feeding through an enteral tube.

6351 Biscayne Blvd.

REFERENCES

1. Bland, J. H.: Clinical use of fluid and electrolyte. W. B. Saunders Co., Philadelphia, 1952.

2. Darrow, D. C., and Pratt, E. L.: Fluid therapy: relation to tissue composition and expenditure of water and electrolyte. J. Am. M. A. 143:365-373, May 27, 1950; 432-439, June 3, 1950.

3. Everett, M. R.: Medical biochemistry. Ed. 2. Paul B. Hoeber, New York, 1946.

4. Elman, R.: Parenteral alimentation in surgery with special reference to proteins and amino acids. Paul B. Hoeber, New York, 1947.

5. Elman, R.: Surgical care. Appleton-Century-Crofts Co., New York, 1951.

6. Mason, R. L., and Zintel, H. A. eds.: Preoperative and postoperative treatment. Ed. 2. W. B. Saunders Co., Philadelphia, 1946.

7. Drew, C. R., Scudder, J., and Papps, J.: Controlled fluid therapy with hematocrit, specific gravity, and plasma protein determination. Surg., Gynec. & Obst. 70:859-867, May 1940.

8. Abbott, W. E.: Fluid, electrolyte and nutritional problems in surgery. S. Clin. North America 26:1330-1360, Dec. 1946.

9. Berger, E. Y., *et al.*: Body water compartments in man. Federation Proc. 8:10, March 1949.

10. Berger, E. Y., and Steele, J. M.: Electrolyte and water metabolism; physiologic considerations. M. Clin. North America 36:829-846, May 1952.

11. Deane, N.: Intracellular water in man. J. Clin. Investigation 30:1469-1470, Dec. (pt. 2) 1951.

12. Edelman, I. S.: Body composition: studies in human being by dilution principle. Science 115:447-454, April 25, 1952.

13. Gamble, J. L.: Chemical anatomy, physiology and pathology of extracellular fluid. Ed. 5. Harvard University Press, Cambridge, Mass., 1947.

14. Harper, H. A.: Review of physiological chemistry. Ed. 3. Univ. Med. Pub., Palo Alto, Calif., 1951.

15. Levitt, M. F., and Gaudino, M.: Measurement of body water compartments. Am. J. Med. 9:208-215, Aug. 1950.

16. Soberman, R., *et al.*: Use of antipyrine in measurement of total body water in man. J. Biol. Chem. 179:31-42, May 1949.

17. Butler, A. M., and Talbot, N. B.: Parenteral-fluid therapy; estimation and provision of daily maintenance requirements. New England J. Med. 231:585-590, Oct. 26, 1944.

18. Butler, A. M., and Talbot, N. B.: Parenteral-fluid therapy; estimation of losses incident to starvation and dehydration with acidosis or alkalosis and provision of repair therapy. New England J. Med. 231: 621-628, Nov. 2, 1944.

19. Barrow, D. C.: Body-fluid physiology; relation of tissue composition to problems of water and electrolyte balance. New England J. Med. 233:91-97, July 26, 1945.

20. Randall, H. T.: Water and electrolyte balance in surgery. S. Clin. North America 32:445-469, April 1952.

21. Bodansky, O.: Parenteral fluid therapy. M. Clin. North America 34:395-412, March 1950.

22. Moyer, C. A.: Fluid balance. Year Book Publishers, Chicago, 1952.

23. Weisberg, N. F.: Water, electrolyte and acid-base balance. Williams & Wilkins Co., Baltimore, 1953.

24. Gastineau, C. F.: Classification of disorders of electrolyte metabolism. Proc. Staff Meet., Mayo Clin. 25:630-634, Nov. 8, 1950.

25. Rosenheim, M. L.: Sodium. Lancet 2:505-511, Sept. 22, 1951.

26. Butler, A. M.: Parenteral fluid therapy in diabetic coma. Acta paediat. 38:59-70, 1949.

27. Gaspar, M. R.: Potassium problem in surgery. Am. Surgeon 18:524-530, May 1952.

28. Elman, R., *et al.*: Intracellular and extracellular potassium deficits in surgical patients. Ann. Surg. 136:111-131, July 1952.

29. Webster, D. R., Henrikson, H. W., and Currie, D. J.: Effect of potassium deficiency on intestinal motility and gastric secretion. Ann. Surg. 132:779-785, Oct. 1950.

30. Lans, H. S., Stein, I. F., Jr., and Meyer, K. A.: Diagnosis, treatment, and prophylaxis of potassium deficiency in surgical patients; analysis of 404 cases. Surg., Gynec. and Obst. 95:321-330, Sept. 1952.

31. Finch, C. A., Sawyer, C. G., and Flynn, J. M.: Clinical syndrome of potassium intoxication. Am. J. Med. 1:337-352, Oct. 1946.

32. Beattie, J.: Metabolic disturbances after injury. Brit. M. J. 2:813-817, Nov. 22, 1947.

33. Committee on Food and Nutrition, National Research Council: Recommended allowances for various dietary essentials. J. Am. Dietet. A. 17:565-567, June-July 1941.

34. Weichselbaum, T. E., Elman, R., and Lund, R. H.: Comparative utilization of fructose and glucose given intravenously. Proc. Soc. Exper. Biol. & Med. 75:816-818, Dec. 1950.

35. Lockhart, C. E., and Elman, R.: Effect of intravenous glucose and amino acids on glycosuria and urinary output in humans. Surg., Gynec. & Obst. 88:97-102, Jan. 1949.

36. The adrenal in protein and carbohydrate metabolism. Nutrition Rev. 9:173-176, June 1951.

37. Wyshak, G. H., and Chaikoff, I. L.: Metabolic defects in liver of fasted rats as shown by utilization of C^{14}-labeled glucose and fructose. J. Biol. Chem. 200:851-857, Feb. 1953.

38. Miller, M., *et al.*: Metabolism of intravenous fructose and glucose in normal and diabetic subjects. J. Clin. Investigation 31:115-125, Jan. 1952.

39. Craig, J. W., *et al.*: Metabolism of fructose by liver of diabetic and non-diabetic subjects. Proc. Soc. Exper. Biol. & Med. 78:698-702, Dec. 1951.

40. Rice, C. O., Orr, B., and Enquist, I.: Parenteral nutrition in surgical patient as provided from glucose, amino acids and alcohol; role played by alcohol. Ann. Surg. 131:289-306, March 1950.

41. Newman, H. W., Wilson, R. H. L., and Newman, E. J.: Direct determination of maximal daily metabolism of alcohol. Science 116:328-329, Sept. 26, 1952.

42. Shafiroff, B. G. P., *et al.*: Subcutaneous administration of combined fat emulsion with hyaluronidase. Proc. Soc. Exper. Biol. & Med. 77:608-611, Aug. 1951.

43. Van Itallie, T. B., Moore, F. D., and Stare, F. J.: Oral fat emulsions in study of calorie-nitrogen relationships in man. Ann. New York Acad. Sc. 56:28-36, Oct. 10, 1952.

44. Sherman, H. C.: Chemistry of food and nutrition. Ed. 5. Macmillan Co., New York, 1937.

45. Elliott, J., *et al.*: New regimen in feeding critically ill; preliminary report. Arch. Surg. 64:278-285, March 1952.

46. Elman, R.: Time factor in retention of nitrogen after intravenous injection of mixture of amino-acids. Proc. Soc. Exper. Biol. & Med. 40:484-487, March 1939.

47. Cannon, P. R., *et al.*: Influence of time of ingestion of essential amino acids upon utilization in tissue-synthesis. Federation Proc. 6:390, March 1947.

48. Ravdin, I. S., and Gimbel, N. S.: Protein metabolism in surgical patients. J. Am. M. A. 144:979-982, Nov. 18, 1950.

Diagnosis of Pancreatitis

M. T. GUTENSOHN, D.O.
Kirksville, Mo.

The diagnosis of pancreatitis is at times very baffling and challenging inasmuch as it is a fairly frequent disease and is quite often misdiagnosed as some other abdominal dysfunction. It is possible that it is much more common than is usually believed, and some of its manifestations may not always be apparent. Perhaps the reason for some of this confusion is the fact that the etiology of both acute and chronic pancreatitis is controversial and somewhat obscure. The etiologic factors in acute pancreatitis are as follows:[1]

1. Infectious pancreatitis
   a. Lymphogenous
   b. Hematogenous
   c. Extension from duodenum or bile ducts via pancreatic ducts
   d. Direct extension from foci of infection or adjacent diseased organs
   e. Activation of enzymes by bacteria in normal gland
2. Noninfectious pancreatitis
   a. Due to back flow into the pancreatic duct of:
      (1) Bile ("common channel" therapy)—incidental to stone or edema of ampulla of Vater, spasm of sphincter of Oddi, or miscellaneous factors
      (2) Duodenal contents
   b. Pancreatic duct obstruction by:
      (1) Epithelial metaplasia
      (2) Stone in pancreatic duct or ampulla of Vater
      (3) Tumor, stricture, or edema
      (4) Duodenal diverticulum.
   c. Trauma
   d. Vascular accidents: embolus, thrombus, rupture
3. Any combination of factors.[1]

The above factors are thought to be causative agents in pancreatitis, but whether or not this list is complete is not as yet known.

Insofar as the clinician is concerned he is usually presented with three main forms of pancreatitis, acute hemorrhagic pancreatitis, acute pancreatic edema, and chronic relapsing pancreatitis.

ACUTE HEMORRHAGIC PANCREATITIS

This is an acute abdominal disease that is serious and sometimes fatal, It has been recognized for many years that it is necessary to differentiate acute pancreatitis from other acute abdominal crises that may need immediate surgery. It may mimic so many acute diseases necessitating surgical procedures that it is very difficult to recognize and use only conservative management. The classic case presents itself in an individual who, usually after a heavy meal or a drinking bout, has been stricken very suddenly with epigastric pain, often somewhat bandlike, across the upper abdomen and radiating into the back. Shock is often present early. Usually the pulse is fast, the temperature low, and the upper abdomen acutely tender with not as much rigidity as would be expected with the amount of pain present. However, the upper abdomen may be almost boardlike while the lower abdomen may be flaccid or only tender to palpation. The onset is usually acute, but it may come on with only mild epigastric discomfort and progress to severe pain. The pain may be either in the upper right quadrant if the pancreatic head alone is involved, in the epigastrium if the body is involved, or it may be left-sided pain which is occasionally referred to the left hypochondrium if the tail of the pancreas is the most affected; if the whole pancreas is edematous the pain will be bandlike and boring, radiating into the back.[2] The patient appears to be quite ill; nausea and vomiting and distention of the abdomen occur in most cases, and at times the patient is in shock. Some writers[1] have mentioned an early bluish mottled appearance of the skin of the limbs which is probably due to the shock. Peristalsis will be absent, or only a few sounds will be heard on auscultation of the abdomen. On palpation of the back there will be an area of perivertebral tenderness and rigidity from the sixth to the tenth or eleventh thoracic vertebrae. On x-ray survey of the abdomen in an acutely ill patient, it is not uncommon to find a sentinel loop of distended jejunum or early paralytic ileus. In the patient who is not too ill to ingest barium, a wide duodenal curve, or "C," from extrinsic pressure on the duodenum by the head of the pancreas will probably be found.

Laboratory tests that have diagnostic significance are not many; however, there are a few that are very important in the diagnosis of early pancreatic inflammatory change. The serum amylase and serum lipase are perhaps the two best diagnostic tests available. The serum amylase is elevated very early in the course of

the disease, but may return to normal in the first 24-hour period, and at the end of 72 hours usually be within normal limits. The test can be performed in 1 hour and should be done in every case of acute upper abdominal pain in which the diagnosis is at all in doubt. The serum lipase test takes 24 hours and, therefore, is not as good a diagnostic test as the serum amylase. The white blood count and sedimentation rate are generally high, and there is quite an increase in the polymorphonuclear cells. The urine is usually concentrated, and there may be glycosuria. Hyperglycemia may be present in the blood, and there may be a subnormal prothrombin level. A test that is of some diagnostic significance is Loewi's test which consists of instilling 3 drops of 1:1,000 epinephrine solution in the patient's eye, followed in 5 minutes by instillation of 3 more drops. Ordinarily the normal eye does not react to epinephrine, but in patients with acute pancreatitis the pupils dilate.[3]

In hemorrhagic pancreatitis there may be free bleeding into the abdominal cavity, and in these cases there may be a bluish-like discoloration around the umbilicus due to blood (Cullen's sign).[4] There may also be a bluish-gray discoloration of the flank which is known as Grey-Turner's sign. Aspiration of the left flank for free peritoneal fluid may be of diagnostic value, for if fluid is obtained it will have a high amylase content. A sign that is of diagnostic significance after a few days of illness is the level of the blood calcium. It has been observed in acute pancreatitis that low values occur between the third and fourteenth days, most often about the fourth or fifth day. This is of value in confirming the diagnosis, but it has greater prognostic value. Although a blood calcium level below 9 mg. per 100 cc. is diagnostic, a level below 7 mg. has serious prognostic implications. A serum calcium level below 7 mg. per 100 cc. usually indicates a very severe form of the disease and a very grave prognosis.

One symptom that is not utilized as much as it could be is the disorientation, inability to recognize surroundings or intimates, or hallucinations which occur in many patients with acute upper abdominal pain due to acute pancreatitis.[5] These findings are very seldom present in any other acute upper abdominal disease. The exact causative agent of central nervous system signs is not known, but they may be due to bloodborne lipase attacking the central nervous system which is high in fat content, or to the absorption of partially digested proteolytic substances from the inflamed pancreas.

The early diagnostic findings are:

1. Severe epigastric pain
2. Elevation of blood amylase
3. Hallucinations or disorientation
4. Subnormal prothrombin level
5. A high white count and high sedimentation rate
6. X-ray findings of paralytic ileus or a loop of distended jejunum
7. Usually low temperature and shock
8. Glycosuria and hyperglycemia
9. Loewi's sign.

The late diagnostic findings are:

1. Subnormal blood calcium
2. Periumbilical bluish-black discoloration (Cullen's sign)
3. Bluish-gray discoloration of the flank (Grey-Turner's sign).

ACUTE PANCREATIC EDEMA

This is an acute form of pancreatitis in which there is suddenly developing edema and inflammatory change which does not lead to hemorrhage or necrosis. The symptoms are essentially the same as those of acute hemorrhagic pancreatitis except they are less severe, and the patient is not as critically ill. These patients may or may not consult a physician; however, serum amylase will uniformly be high early in the course of the disease. In this type of inflammatory disease of the pancreas conservative management is all that is necessary, and there may be no findings other than the elevated serum amylase, Loewi's sign, and elevation of the white count and sedimentation rate in the presence of acute pain in the abdomen following dietary or alcoholic indiscretion.

CHRONIC PANCREATITIS

Chronic pancreatitis, or, as it is sometimes known, chronic relapsing pancreatitis, is a fairly well known but little understood clinical entity. Chronic alcoholism is present in about 50 per cent of the patients, and many episodes of chronic pancreatitis are associated either with alcoholic or dietary indiscretions. The careful evaluation of the patient's history, the presenting complaint, and physical examination will usually rule out peptic ulcer, cholecystic disease, coronary insufficiency, or functional diseases of the gastrointestinal tract. If these conditions can all be eliminated, chronic relapsing pancreatitis is probably the most likely cause of recurring upper abdominal pain. If it is an extremely acute attack, all of the symptoms of acute hemorrhagic pancreatitis, which have already been enumerated, will be found. However, ordinarily the patient with chronic pancreatitis will present himself with the complaint of upper abdominal distress and probably will not be seen during an acute episode.

Pain is the most common symptom and is usually felt across the whole upper abdomen, radiating to the spine or flank at about the same level as the pain anteriorly. The pain is made worse by alcohol or may come on 1 or 2 hours after a meal. Fasting will usually relieve the pain. In most of these cases, the pain exhibited by the patient cannot be elicited by the examiner, and if tenderness is found on palpation it will extend across the upper abdomen into the flank.

Stool examination may show steatorrhea. There may be diarrhea with bulky and greasy stools, or diarrhea may be completely absent and the excess in fat and protein may only be seen microscopically if at all. This must be differentiated from sprue.

In quite a number of these patients calcification of the pancreas will show on a flat x-ray abdominal survey. It may be single or multiple, and it may be limited to one part of the organ or be widespread. There will probably be enlargement of the duodenal curve by barium ingestion and x-ray.

These patients may exhibit signs of the fat-soluble vitamin deficiencies, especially vitamin A.

The serum amylase may be normal if determined routinely, but if serial levels are used, elevation of the serum amylase can almost always be found.[6] It may be necessary to stimulate amylase and lipase by using intravenous secretin.[7]

An irregular, tender, and elongated mass may be palpated in the upper epigastrium. Many of these patients will have glycosuria and hyperglycemia which are due to destruction of the islets of the pancreas by continuously recurring inflammatory change. If there

is diabetes, it will be very difficult to control as it is of the brittle or labile type. If enough insulin is given to keep diabetic patients sugar free, they are very apt to have frequent attacks of insulin shock. A glucose tolerance test will be either normal or diabetic in type.

Weight loss is a very common finding in these patients due either to the alcoholic abuse or to the fact that they have found out that fasting will usually keep them from having the pain. In addition they are very apt to eat an unbalanced diet.

Diagnostic findings are:

1. Recurrent epigastric pain
2. Weight loss
3. Hyperglycemia and glycosuria
4. X-ray indication of calcification and enlargement of the duodenal curve
5. Marked variation in serum amylase determination.

SUMMARY

1. Known etiologic factors of pancreatitis are listed.

2. Diagnostic findings of pancreatitis are discussed.

3. An elevation of the serum amylase appears to be the most diagnostic test in all types of pancreatitis.

Box 898

REFERENCES

1. Dozzi, D. L., and Bockus, H. L.: Acute pancreatitis, pancreatic injuries and pancreatic fistula, in Gastro-enterology, by H. L. Bockus, *et al.* W. B. Saunders Co., Philadelphia, 1946, vol. III, pp. 769-791.

2. Zollinger, R. M.: Pancreatitis. Postgrad. Med. 15:323-333, April 1954.

3. Goodman, L., and Gilman, A.: Pharmacological basis of therapeutics. Macmillan Co., New York, 1941.

4. Pullen, R. L.: Medical diagnosis. Ed. 2. W. B. Saunders Co., Philadelphia, 1950.

5. Garrett, S. Y.: Etiology, pathologic physiology and diagnosis of acute pancreatitis. Am. Pract. & Digest Treat. 3:33-38, Jan. 1952.

6. Logan, V. W.: Acute and chronic pancreatitis. Postgrad. Med. 13:261-266, March 1953.

7. Lopusniak, M. S., and Bockus, H. L.: Study of pancreatic serum enzymes following secretin injections of pancreatic affections. Gastroenterology 16: 294-308, Oct. 1950.

The Diagnosis of Infectious Hepatitis

ROBERT SEDAR, D.O.
Denver

The term "infectious hepatitis" in this paper refers to the disease process caused by the hepatotrophic filtrable virus identified by laboratory methods and known as the I.H. virus.[1] No reference will be made to the closely related clinical entity, serum hepatitis, caused by the laboratory identifiable S.H. virus. The discussion will concern itself with diagnosis based upon the following classification: (1) preicteric stage or prodromal phase, (2) icteric stage or acute phase, and (3) posticteric stage or chronic phase.

PREICTERIC STAGE

Accurate diagnosis during the prodromal phase of any disease depends largely upon the intellectual curiosity of the clinician, selective laboratory testing, and careful anamnesis. This is especially true in infectious hepatitis. During the preicteric stage and in the nonicteric variety, manifestations may mimic any number of acute abdominal conditions. The astute clinician will do well to place in a "rule-out category" all endemic infectious cases occurring somewhat seasonally in the fall and early winter which involve children and young adults. It is well to remember that the geographic distribution is world-wide.

The symptomatology is extremely variable. Abrupt temperature elevations ranging from 100 to 104 F. usually usher in the disease. Persistent anorexia, one of the most significant symptoms, is followed by malaise, fatigue, cephalalgia, chills, nausea, emesis, pruritus, amber-colored urine, alterations in color and character of stools, and upper right quadrant distress. These signs and symptoms justify investigation to determine the exact nature of the disease. Only too often these symptoms are attributed to "grippe," "intestinal flu," "gastroenteritis," "virus x," and "biliary dyskinesia" without the benefit of additional investigational procedures.

It has been estimated that from two to eight unrecognized cases without jaundice occur for every case with jaundice. As there is no specific diagnostic procedure, an accurate definition of the total incidence is not ascertainable.

The usual incubation period of infectious hepatitis ranges from 2 to 6 weeks. It is here that a carefully obtained history may reveal a possible known contact or a situation which might plausibly have resulted in inoculation.

Physical examination may or may not be revealing at this time. The liver may be slightly enlarged and tender upon palpation. The fist percussion test may be positive, that is pain occurs within 5 seconds of percussion and persists for several hours.[2] Unusual exercise intolerance may produce a "liver ache" which may last for days. In endemics, mild generalized lymphadenopathy of the posterior-inferior cervical chains may be observed. Changes in the sclera and mucous membrane should be anticipated.

As previously stated, there is no specific diagnostic procedure in this disease. Therefore, the laboratory must be relied on to point out altered liver function. Only too often laboratory values may be entirely normal. This may occur in spite of some ninety-seven liver-function tests devised to calculate seventy-seven liver functions involving some 5,000 chemical reactions.[3] However, elevation of urinary urobilinogen and serum alkaline phosphatase and Bromsulphalein retention may precede the appearance of jaundice.

Bilirubinuria in morning specimens often appears prior to jaundice.[1] The cephalin flocculation test must be more than 2+ to be considered positive in hepatocellular dysfunction. In instances of reduced serum albumin, whether from an extrinsic or intrinsic defect, the cephalin flocculation test will be falsely positive owing to the lack of sufficient serum albumin to inhibit the flocculation of cephalin-cholesterol.[4] Thus it would appear that this is not a true test of liver function.

The importance of early diagnosis is from two diverse standpoints. First, the patient is more likely to

benefit from early, more vigorous supportive care, perhaps thereby minimizing the manifest sequela, chronic hepatitis. Secondly, from an epidemiologic viewpoint, the incidence of secondary attack rates may be reduced by employing gamma globulin as early prophylaxis. Recent reports seem to indicate that this is a very effective method. In families exposed to infectious hepatitis, administration of gamma globulin reduced the secondary attack rate to 0.6 per cent. In control families where no gamma globulin was administered, the secondary attack rate was 15.2 per cent.[5] Thus it appears that early diagnosis and prophylactic use of gamma globulin will do much to limit endemic spread. The estimated effective dosage of gamma globulin is now set at 0.01 cc. per pound of body weight.[6,7]

The duration of the preicteric stage averages 6 days, but may range from a few hours to 3 weeks.[1] While jaundice is usually an early manifestation of severe hepatitis, death may occur before its appearance in fulminating cases.

ICTERIC STAGE

The diagnostic criteria during this phase are largely devoted to differentiating between intrahepatic and extrahepatic jaundice. Intrahepatic jaundice may be hemolytic or hepatocellular. In extrahepatic jaundice, consideration must be given to whether the obstruction of bile flow is biliary or extrabiliary; here again the clinician must rely heavily upon the laboratory for a proper differentiation.

In order to differentiate hemolytic from hepatocellular disease, serum bilirubin concentrations should be ascertained. It should be routine to determine the total and the direct and indirect reactions (van den Bergh test). If less than 0.2 mg. per 100 cc. of the bilirubin reacts directly, and the remainder reacts indirectly, it is suggestive of jaundice attributable to a hemolytic process or to constitutional hepatic dysfunction.[4] The performance of the 24-hour fraction of the 72-hour urobilinogen in the stool will serve to differentiate these intrahepatic conditions. The finding of more than 300 mg. per 100 cc. stool urobilinogen in 24 hours precludes a hemolytic process. Normal values of 40 to 280 mg. in 24 hours will be found in constitutional hepatic dysfunction. If the serum contains more than 0.2 mg. per 100 cc. of direct reacting bilirubin, either a hepatocellular or obstructive phenomenon exists.[4] A total serum bilirubin greater than 10 mg. per 100 cc. indicates complete common duct obstruction.

Often apparent acholia occurs, which only makes a perplexing problem more complex. It is a usual finding in obstructive disease. It is well to remember that it is not unusual to note absence of bile in the stool in cases of hepatocellular disease, but the absence is only for transient periods. By repeated duodenal drainage, which may be necessary over a period of 6 to 10 days, the appearance of bile is detected, thus indicating common duct patency.

Upon excluding the possibility of hemolytic disease and complete obstruction, it is still necessary to further differentiate partial obstruction from true hepatic parenchymal involvement. If the serum alkaline phosphatase, an excretory product of the hepar, is greater than 12 Bodansky units, biliary obstruction is indicated. Prothrombin time response to oral water-soluble vitamin K, in instances of previously decreased prothrombin time, suggests an obstructive type of jaundice.[4] Four mg. of water-soluble vitamin K taken 24 hours earlier are sufficient for the test. Elevated total serum cholesterol, in the absence of signs of hypometabolism, diabetes mellitus, and exogenous dietary excess, would tend to indicate obstructive disease. If the ester fraction of serum cholesterol is reduced below its normal value of approximately 66 per cent of the total, a severe hepatocellular alteration is in effect. Estimation of the total serum proteins coupled with the albumin-globulin ratio may aid in evaluating the hepatic status. Normal values for total serum protein range from 6.5 grams to 8 grams per 100 cc., and approximately 60 to 65 per cent of this total should be represented by the serum albumin. A decreased total protein coupled with a reversed albumin-globulin ratio should serve to confirm altered hepatocellular physiology.

Unfortunately, it is not always possible to establish hepatic disease conclusively through the laboratory medium. While adequate clinical and laboratory methods usually distinguish primary parenchymatous from extrahepatic disease in the jaundiced patient, there still remains a high percentage in which an exact clinical diagnosis cannot be made. It is here that the use of the needle biopsy must be given consideration.

Much has been written both for and against the needle biopsy technic. However, in the hands of experienced operators, under selected conditions, it remains a very effective diagnostic modality. Zamcheck and Klausenstock,[8] in a recent review of the literature, reported only thirty-four deaths directly attributed to the procedure in 20,016 needle biopsies, a mortality of 0.17 per cent. Thus it would seem that the procedure is not unduly dangerous.

The technic is relatively simple. The first step is to give a full explanation of the procedure to the patient, emphasizing the need for complete cooperation and the absolute necessity for remaining motionless. Practice sessions in breath holding may lessen difficulties. After a mild sedative is administered preoperatively with a suitable agent such as Meperidine hydrochloride, the procedure is inaugurated. Local infiltration anesthesia is accomplished in one of two commonly employed anatomic sites. With a Roth-Turkel needle especially designed for the purpose, a satisfactory liver core can be obtained for histopathologic analysis. The most common approach, used when the liver margin is palpable, is through the anterior abdominal wall below the right costal margin. The second site, used with the right transthoracic approach, through the right lateral chest wall, is relegated to cases of small or nonpalpable livers.[9] In many instances, by this means of analysis of tissue sufficient evidence is afforded to establish diagnosis.

Clinically, the course of the disease progresses with deepening jaundice, protracted nausea with emesis, and extreme debility. The symptoms are intensified at first; however, the patient is usually on his way to recovery with the return of the appetite. This stage may last from 2 to 4 weeks, providing the disease does not persist into the chronic phase.

POSTICTERIC STAGE

The third and final phase is found in those cases which have persisted for at least 6 months after the onset of the initial symptoms of the disease. An accurate history may reveal a previous hepatitis syndrome of the nonicteric variety. In epidemics the nonicteric incidence has been reported to range from 28 to 80 per cent of the total number of infections.[1]

The severity of the disease may range from cases in which the patient is asymptomatic and the laboratory and biopsy findings minimal, to those in which the patient is physically incapacitated owing to postnecrotic cirrhosis.

Frequent symptoms are persistent liver pain, anorexia with peculiar protein intolerance, fatigability, nausea, with or without emesis, malaise, and vague gastrointestinal complaints.

Physical examination reveals emaciation; increased capillary fragility, measured by the Rumpel-Leede tourniquet test; liver enlargement with tenderness; spider angiomata; palmar erythema; and splenomegaly. Various degrees of icterus are encountered, most noticeably in the sclera and on the skin of the abdomen.

The laboratory findings are usually more conclusive than either the symptomatology or physical findings. Tests usually positive are the Bromsulphalein retention, with a range from 5 to 20 per cent, urine urobilinogen, zinc sulfate turbidity, and thymol turbidity.

Unfortunately, a large percentage of cases, as high as 17 per cent, may have no positive laboratory findings. In these instances, liver biopsy by the needle or the laparotomy technic will reveal characteristic mononuclear infiltration and fibrosis and provide the only confirmation.

Repeated needle biopsy, at varying sites, often is an aid in determining progressive or regressive parenchymal changes. This can provide an excellent means of determining the efficacy of treatment.

CONCLUSION

The diagnosis of infectious hepatitis offers a constant challenge to the clinician. Even with advancements and refinements in clinical and laboratory methods there is still room for improvement. A ray of hope appears on the diagnostic horizon with the recent announcement by the Atomic Energy Commission which predicts over 90 per cent accuracy in the diagnosis of liver dysfunction with the use of radioactive isotopes.[10,11]

3300 East 17th Avenue

REFERENCES

1. Cecil, R. L., and Loeb, R. F., eds.: Textbook of medicine. Ed. 8. W. B. Saunders Co., Philadelphia, 1951.
2. Harrison, T. R., ed.: Principles of internal medicine. Blakiston Co., Philadelphia, 1950.
3. Knisely, N. H.: Discussion of Serum proteins and lipids. R. H. Turner, in Transactions of Tenth Conference on Liver Injury, edited by F. W. Hoffbauer. Josiah Macy, Jr., Foundation, New York, 1951, pp. 47-52.
4. Cain, J. C.: Differential diagnosis of mild, painless jaundice. Collect. Papers Mayo Clin. & Mayo Found. 45:81-87, 1953.
5. Ashley, A.: Gamma globulin; effect on secondary attack rates in infectious hepatitis. New England J. Med. 250:412-417, March 11, 1954.
6. Hsia, D. Y., Lonsway, M., Jr., and Gellis, S. S.: Gamma globulin in prevention of infectious hepatitis; studies on use of small doses in family outbreaks. New England J. Med. 250:417-419, March 11, 1954.
7. State of Colorado, Department of Health Bulletin, June 18, 1954.
8. Zamcheck, N., and Klausenstock, O.: Liver biopsy. II. Risk of needle biopsy. New England J. Med. 249:1062-1069, Dec. 24, 1953.
9. Zamcheck, N., and Sidman, R. L.: Needle biopsy of liver. I. Its use in clinical and investigative medicine. New England J. Med. 249: 1020-1029, Dec. 17, 1953.
10. Personal communication. Abbott Laboratories, Chicago, 1954.
11. MacIntyre, W. J. *et al.*: ^{131}I-labeled serum albumin; its use in study of cardiac output and peripheral vascular flow. Radiology 59:849-857, Dec. 1952.

The Nephrotic Syndrome in Children

LEO C. WAGNER, D.O.
Professor of Pediatrics
Philadelphia College of Osteopathy
Philadelphia

Until more is known of the causes and mechanisms involved in the nephrotic syndrome, evaluation of prognosis must be statistical, treatment can only be symptomatic, and prevention is impossible. The purpose of this discussion is to assemble the current etiologic, pathologic, physiologic, and clinical knowledge of the nephrotic syndrome.

By many internists this syndrome is considered a form of chronic glomerulonephritis with an unrecognized onset, clearly related to acute nephritis. Pediatricians, on the other hand, consider it a single distinct entity not preceded by unrecognized acute glomerulonephritis but sometimes associated with signs of nephritis. Some of the reasons for the latter view are:

1. It is well established that 1 to 3 weeks before its occurrence acute glomerulonephritis is preceded by a rather severe infection, usually upper respiratory, and in most instances it is caused by hemolytic streptococcus as shown by pure cultural growths and by increase in antistreptolysin titers, whereas in the nephrotic patient there is no history of an antecedent infection and the antistreptolysin titers are always below normal.

2. The generally accepted hypothesis that acute glomerulonephritis is an antigen-antibody phenomenon instigated in the kidney in some fashion is based on the following: (a) the onset is preceded by a streptococcal infection, (b) there is a latent period between the time of infection and the initial onset of acute symptoms, and (c) there is a more immediate response of the kidney to subsequent streptococcal infections in chronic cases. On the other hand, in the nephrotic patient such analogous relationships to infections are not so clearly defined.

3. Superimposed acute infections in acute nephritis are followed by exacerbation of the renal symptoms, whereas in the nephrotic syndrome there is a greater frequency of remission of symptoms.

There is general agreement that the nephrotic syndrome consists of five cardinal entities, proteinuria, hypoproteinemia, hypoalbuminemia, hypercholesterolemia, and edema, but there is not agreement as to their cause.

Several hypotheses have been advanced to explain the proteinuria which is so characteristic of nephrosis. The most common are that (1) it is due to failure of tubular resorption of normally filtered proteins, (2) abnormal plasma proteins are formed which pass more

readily through the glomerular wall, and (3) the glomerular wall shows increased permeability to proteins. Barnett and his associates[1] have presented numerous reasons why the first two hypotheses are not tenable while the third adequately explains the abnormality.

At one time hypothyroidism was thought to be an etiological factor because of the hypercholesterolemia which is always associated with nephrosis. However, the following facts argue against the role of the thyroid in the nephrotic syndrome: (1) When the metabolic rate is calculated on the ideal weight rather than the actual weight, which includes edematous fluid, it is found that the metabolic rate is not abnormally low. (2) Large doses of thyroid substances are ineffectual therapeutically. (3) Most of the clinical signs of hypothyroidism are absent. (4) Hypercholesterolemia in hypothyroidism is not accompanied by an increase in neutral fats as it is in the nephrotic syndrome.[2]

The protein-bound iodine is as low in some patients with nephrosis as in patients with severe myxedema, but it is generally believed that most, if not all, of the protein-bound iodine is more or less loosely bound to the albumin of the serum proteins.[3,4] Consequently, the low protein-bound iodine can be directly attributed to the proteinuria rather than to the hypothyroidism.

While the mechanisms for hyperlipemia are still unknown, some interesting data relative to lipemia have been uncovered. In the average normal subject 29 per cent of the total cholesterol (54 mg. per 100 cc.) is associated with Fraction A of the plasma protein (which contains most of the alpha-lipoproteins) and 64 per cent (123 mg. per 100 cc.) with Fraction C (which contains most of the beta-lipoproteins), whereas in the nephrotic patient only about 5 per cent (27 mg. per 100 cc.) is contained in Fraction A and 92 per cent (539 mg. per 100 cc.) is associated with Fraction C. The total cholesterol averages 577 mg. per 100 cc. in the nephrotic patient and 190 mg. per 100 cc. in the normal subject.[5,6] The cause of the changes is still to be ascertained.

The pathogenesis of edema in the nephrotic syndrome is complex, but edema is ultimately due to excessive retention of salt and water by the kidneys. Insufficiency in the glomerular filtration rate relative to the rate of tubular reabsorption of sodium, chloride, and water appears to play a major role. Excess of posterior pituitary antidiuretic hormone may play a part, also.

Farr[7] has aptly said that to the child with the disease and to his parents the nephrotic syndrome is edema. Except in rare instances it is the appearance of edema which leads to the examination of the urine and the diagnosis of the nephrotic syndrome. It occurs most frequently in children between 2 and 5 years of age who invariably have had an antecedent history of good health. The low incidence of preceding infections and the absence of seasonal incidence is in striking contrast to the histories of children with acute glomerulonephritis.

Although edema may be present constantly, periods of varying but usually severe degrees of edema alternating with remissions during which edema is completely absent are more characteristic.

The onset of remissions, one or more of which occur in approximately 95 per cent of patients, is usually marked by abrupt, profuse, and sustained diuresis, which may occur spontaneously or follow infections. The remissions, or subsequent exacerbations, may last for weeks or months, and although the disease may last for as little as 2 weeks,[8] it is usually measured in months or years.

Increased susceptibility to infections, especially severe pneumococcal infections, is a constant threat. Such infections are characterized by sudden elevation of temperature, abdominal or flank pain (usually less tenderness than is expected in peritonitis), vomiting, diarrhea, and an erysipeloid eruption on the abdomen or thighs. These episodes have been designated nephrotic crises and are considered by some authorities as metabolic disturbances which are usually but not always associated with infections. However, many others believe such crises to be due to infections even though this is not proved by positive cultures.

These children have notoriously poor appetites and this, added to the great loss of protein in the urine, results in a severe degree of malnutrition which is frequently concealed by the edema.

Gastrointestinal disturbances not associated with infections are frequently encountered. Diarrhea is especially common during periods of edema and has been attributed to edema of the intestinal mucosa.

Respiratory difficulty resulting from abdominal distention with or without pleural effusion may be disturbing and occasionally alarming. In short, most of the physical abnormalities are related to the edema: Minor skin irritation around the genitals, eyes, neck, et cetera, results from friction or pressure of edematous parts; prominence of the anterior abdominal wall veins results from abdominal ascites; rectal prolapse is not uncommon and also results from edema.

From a diagnostic point of view, it is interesting that of the five cardinal signs of the nephrotic syndrome, four represent changes that can be determined by laboratory examinations. Barnett and his associates[1] compared the laboratory findings of twenty-five cases of nephrotic syndrome with those of twenty-five cases of acute glomerulonephritis with the following results:

	Nephrotic syndrome	Acute glomerulonephritis
Total Protein gm/100 cc.	4.0 ± .5	6.6 ± .8
Albumin gm/100 cc.	1.7 ± .4	4.2 ± .7
Urea Nitrogen mg/100 cc.	18.9 ± 4.1	27.7 ± 4.6
Cholesterol mg/100 cc.	730 ± 81	240 ± 39

While the diagnosis of the nephrotic syndrome is clear-cut and definite, the prognosis is difficult and indefinite. The relationship between prognosis and the clinical picture at the onset is difficult to decide from available data. Numerous reports have appeared in the literature showing that some children recover who have normal renal clearances, no hypertension, and no hematuria throughout the disease, and yet some children with the same findings will eventually die of renal insufficiency. On the other hand, some will show reduced clearances, with hypertension and hematuria at the onset and yet recover completely. It is obvious, therefore, that there is no correlation between the number and duration of exacerbations and the ultimate outcome. Even though intercurrent infections have been treated successfully and severe electrolytic imbalance has been avoided, the prognosis depends upon what happens to kidney function.

While the prognosis of the nephrotic syndrome must be vague and unpredictable, it is essential for the parents to know that although edema is important in terms of how the child looks and feels, it does not determine, nor can it be used to judge, the outcome. Such knowledge can help minimize the emotional swings which parents experience with each recognizable increase or decrease in edema.

Perhaps in no other childhood disease is more pressure exerted on the physician to "do something" than in the nephrotic syndrome. This probably arises from the distressing symptoms, uncertain prognosis, and the understandable anxiety of the parents and sometimes of the physician himself, who realizes the inadequacy of available therapy. Treatment designed to reduce edema is of great symptomatic importance, but the real problem concerns treatment directed toward healing the renal lesion. It is intended here not to review the multitude of regimens which have been proposed, but rather to describe the principles of treatment which at present seem most reasonable.

These children should not be hospitalized except at the onset to permit diagnosis and evaluation, in the presence of severe infections or massive edema or ascites producing respiratory embarrassment, and for hormonal therapy.

There has been no convincing evidence from any study that restriction of activity has any influence on the ultimate outcome of the disease. This, plus the extreme difficulty of enforcing complete bed rest on young children, makes restriction of activity a seemingly useless procedure.

High protein diets have been advocated for these patients. However, attempts to force children to eat high protein diets have largely met with failure and, at present, a high protein diet is recommended if the child will accept it; otherwise, the ordinary amounts of protein for the child's age are sufficient. In addition, salt-free diets have been advised even to the use of low sodium milk and low sodium bread. Such restriction not only makes the diet unpalatable and, hence, interferes with the child's appetite, but may precipitate what Schroeder[9] has termed the low salt syndrome and also marked elevation of serum potassium. Therefore, merely avoiding the addition of salt to food seems a more practical rationale.

At present, ACTH appears to be the most satisfactory means of inducing diuresis. Intramuscular injections of 50 to 100 mg. of ACTH per day in four equal doses are given for 10 days.[1] Should diuresis occur during ACTH administration, no further injections are given. Usually diuresis occurs 1 to 4 days after therapy is discontinued. Failure of ACTH therapy to induce diuresis warrants a second course a week to 10 days later. If two courses of ACTH therapy fail to induce diuresis, no further attempt to do so should be made with this drug. However, it may be used at any time that there is a reaccumulation of edema.

Since the use of ACTH is not without danger, it is not given as soon as the diagnosis of nephrosis is made. It is felt that it should be withheld for 6 to 8 weeks following the onset of the disease or an exacerbation in order to permit spontaneous diuresis to occur. When it is decided to use ACTH therapy the child should be hospitalized and carefully studied before the therapy is instituted.

Hypertension, recent infections, marked deviations of serum electrolyte patterns, or uremia are considered contraindications to ACTH therapy.[10] Equally as important are the complications of this treatment, such as hypotonicity of the extracellular fluid accompanied by low serum potassium, alkalosis, and tetany. Furthermore, ACTH administration may mask signs of serious infections. For this reason, prophylactic antibiotic therapy should be used in addition to constant observation. Marked lethargy, irritability, and fever during diuresis suggest an underlying infection and indicate immediate cessation of ACTH therapy with a change or an increase in antibiotic therapy. Cardiovascular complications must also be watched. Progressive elevation of blood pressure or an increase in preexisting mild hypertension may be an indication for cessation of ACTH administration. It is apparent from the above that ACTH therapy is not an innocuous procedure, and as such is not a safe office or home treatment.

Osteopathic management of renal disease resolves itself into osteopathic manipulative treatment directed to the splanchnic region from whence arise the most important vasomotor nerves of the body. The sympathetics, which furnish both vasoconstrictor and vasodilator fibers, go to the kidney as nonmedulated fibers from the celiac ganglion which is supplied by connector fibers from the sixth dorsal to the first lumbar vertebrae but primarily from the tenth, eleventh, and twelfth dorsal.[11]

Pottenger[12] describes increased tonus or spasm of the lumbar muscles with degeneration of the lumbar muscles and subcutaneous tissue as an important diagnostic sign in chronic inflammatory processes involving the renal tissue. Need anything further be said to justify the rationale for osteopathic manipulation in the nephrotic syndrome?

SUMMARY

1. The current etiologic, pathologic, physiologic, and clinical knowledge of the nephrotic syndrome has been reviewed.

2. The internist's and pediatrician's viewpoints of the nephrotic syndrome and the differences between the two have been mentioned.

3. The cardinal signs of the nephrotic syndrome have been discussed.

4. The pathogenesis of edema in the nephrotic syndrome has been stressed.

5. The prognosis and treatment, including ACTH therapy, have been considered.

237 N. Lansdowne Ave.
Lansdowne, Pa.

REFERENCES

1. Barnett, H. L., Forman, C. W., and Lauson, H. D.: Nephrotic syndrome in children, in Advances in pediatrics, edited by S. Z. Levine. Year Book Publishers, Chicago, 1952, vol. V, pp. 53-128.

2. Peters, J. P., and Man, E. B.: Relation of albumin to precipitable iodine of serum. J. Clin. Investigation 27:397-405, July 1948.

3. Taurog, A., and Chaikoff, I. L.: Nature of circulating thyroid hormone. J. Biol. Chem. 176:639-656, Nov. 1948.

4. Salter, W. T.: Metabolic circuit of thyroid hormone. Ann. New York Acad. Sc. 50:358-376, Jan. 27, 1949.

5. Russ, E. M., Eder, H. A., and Barr, D. P.: Protein-lipid relationships in human plasma. I. In normal individuals. Am. J. Med. 11:468-479, Oct. 1951.

6. Barr, D. P., Russ, E. M., and Eder, H. A.: Protein-lipid relationships in human plasma. II. In atherosclerosis and related conditions. Am. J. Med. 11:480-493, Oct. 1951.

7. Farr, L. E.: Effect of dietary protein on urea clearance of children with nephrosis. J. Clin. Investigation. 15:703-710, Nov. 1936.

8. Heymann, W., and Startzman, V.: Lipemic nephrosis. J. Pediat. 28:117-133, Feb. 1946.

9. Schroeder, H. A.: Renal failure associated with low extracellular sodium chloride; low salt syndrome. J. Am. M. A. 141:117-124, Sept. 10, 1949.

10. Metcoff, J., *et al.*: Adrenocorticotrophic hormone (ACTH) therapy of nephrotic syndrome in children. Pediatrics 10:543-564, Nov. 1952.

Advisory Board for Osteopathic Specialists and Boards of Certification

ADVISORY BOARD FOR OSTEOPATHIC SPECIALISTS

Earl E. Congdon, D.O., Chairman
Robert A. Steen, D.O., Secretary

Several specialty boards in osteopathic medicine were in the process of formation prior to 1939 and the demand for others was recognized. Osteopathic specialists and the osteopathic profession felt the need of a single authoritative agency to define graduate education, establish minimum standards that would be basic to such education, furnish information on procedure to the specialty groups, coordinate their efforts, and protect the public by giving validity to certification. To meet these needs, the Advisory Board for Osteopathic Specialists was organized in 1939 by authorization of the Board of Trustees of the American Osteopathic Association. The Advisory Board rendered immediate assistance to existing specialty groups in their efforts to initiate and maintain minimum standards of graduate education against the time when individuals in various specialties would be prepared for certification. It helped in the establishment of additional boards of certification as the growth of specialties within osteopathic medicine demanded.

Certifying boards need assistance in determining the standards of fitness and ability of each applicant for certification as a specialist, they require guidance in defining the scope of the several accepted specialties, and they need advice in establishing standards of education and training for these specialties. An authoritative agency of the Board of Trustees of the American Osteopathic Association is required to maintain a cooperative effort between the certifying boards and educational and training-hospital groups, in order that graduate educational facilities will be constantly available to students in the various specialty fields. By the creation of an advisory board it was possible to obviate duplication of effort, to promote uniformity in the methodology of certification, to prevent unofficial groups from assuming the right of certification, and to inform the profession at large of the significance of specialty certification and its relation to the general practitioner.

The Advisory Board is composed of the secretaries of the approved certifying boards and one other appointed representative from each of the boards, two representatives of the Board of Trustees of the American Osteopathic Association, and representatives from such other national groups as are interested in the education, examination, and certification of osteopathic specialists.

Member organizations comprising the Advisory Board of Osteopathic Specialists are eleven boards of specialty certification, the Board of Trustees of the American Osteopathic Association, the Bureau of Professional Education and Colleges, the Bureau of Hospitals, the American Association of Osteopathic Colleges, and the Central Office of the American Osteopathic Association. The voting membership totals 30.

MEMBERSHIP OF THE ADVISORY BOARD FOR OSTEOPATHIC SPECIALISTS, 1955

BOARD OF TRUSTEES OF THE AMERICAN OSTEOPATHIC ASSOCIATION
Roswell P. Bates — Gus S. Wetzel

BUREAU OF PROFESSIONAL EDUCATION AND COLLEGES
Roy J. Harvey — Reed Speer

BUREAU OF HOSPITALS
Vincent P. Carroll — John P. Schwartz

AMERICAN ASSOCIATION OF OSTEOPATHIC COLLEGES
Wallace M. Pearson — K. J. Davis

CENTRAL OFFICE—AMERICAN OSTEOPATHIC ASSOCIATION
Robert A. Steen

AMERICAN OSTEOPATHIC BOARD OF DERMATOLOGY AND SYPHILOLOGY
Ronald W. MacCorkell — James D. Stover

AMERICAN OSTEOPATHIC BOARD OF INTERNAL MEDICINE
Neil R. Kitchen — Glennard E. Lahrson

AMERICAN OSTEOPATHIC BOARD OF NEUROLOGY AND PSYCHIATRY
Cecil Harris — Thomas J. Meyers

AMERICAN OSTEOPATHIC BOARD OF OBSTETRICS AND GYNECOLOGY
Arthur B. Funnell — Jacquelin Bryson

AMERICAN OSTEOPATHIC BOARD OF OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY
L. A. Lydic — R. S. Licklider

AMERICAN OSTEOPATHIC BOARD OF PATHOLOGY
Robert P. Morhardt — Norman W. Arends

AMERICAN OSTEOPATHIC BOARD OF PEDIATRICS
William S. Spaeth — H. Mayer Dubin

AMERICAN OSTEOPATHIC BOARD OF PHYSICAL MEDICINE AND REHABILITATION
William W. W. Pritchard — Robert C. Ruenitz

AMERICAN OSTEOPATHIC BOARD OF PROCTOLOGY
Carlton M. Noll — Carl S. Stillman, Jr.

AMERICAN OSTEOPATHIC BOARD OF RADIOLOGY
A. H. Witthohn — D. W. Hendrickson

AMERICAN OSTEOPATHIC BOARD OF SURGERY
James M. Eaton — Arthur M. Flack, Jr.

COMMITTEES, 1955

Executive Committee:
Earl E. Congdon, Chairman
Thomas J. Meyers, Vice-Chairman
Robert A. Steen, Secretary
Dorothy J. Marsh
Norman W. Arends

Review Committee (three members):
Appointed annually not later than 30 days before the time of annual meeting.

Appeal Committee (five members):
Appointed when deemed necessary by the Chairman of the Advisory Board with the approval of the Executive Committee.

Committee on Basic Documents:
K. J. Davis
C. M. Noll
Robert Steen

REVISED RULES OF ORGANIZATION AND PROCEDURE

ARTICLE I

Sec. a. The name of this body shall be the Advisory Board for Osteopathic Specialists.

Sec. b. This Advisory Board is constituted and functions under the auspices of the Board of Trustees of the American Osteopathic Association to which it serves as an advisory body and from which it receives authority to engage in those activities necessary to fulfill its purposes. Actions of this Board to have effect must be approved by the Board of Trustees of the American Osteopathic Association.

ARTICLE II—PURPOSES AND DUTIES

The purposes and duties of this Board shall be the following:

Sec. a. To assist in the establishment of certifying boards which shall determine the fitness and ability of each applicant for certification as a specialist in a distinct and recognized specialty.

(1) To be accepted as a certifiable field of specialization such specialty must be accurately definable, must be represented by a reasonable number of individuals who confine themselves or give the preponderance of their time to practice within the defined field; the technics practiced within the field shall be adequate to care for the majority of cases within the field defined; it shall be a field in which knowledge of the entire range of possibilities is a basic qualification and in which only the unusual case calls for special technics not included within the definition and where the practitioner within the field is required to be competent to decide upon the need for special technics.

(2) The definition which is included for the specialty shall be stated so as to delineate accurately the scope of practice properly embraced by the specialty.

Sec. b. To advise the specialty certifying boards with reference to defining and determining the scope of the several accepted specialties.

Sec. c. To advise the specialty certifying boards with reference to establishing appropriate standards of education and training for the several specialties and coordinating the requirements for certification by the certifying boards.

Sec. d. To advise the certifying boards in reference to the development of an appropriate modus operandi for determining fitness of applicants for certification.

Sec. e. To receive from the certifying boards proposals relating to their organization and activities and transmit these with appropriate recommendations to the Board of Trustees of the American Osteopathic Association.

Sec. f. To provide for the certification of applicants in distinct and recognized specialties in which there is no organized specialty society in the profession and no certifying board.

Sec. g. To review the proposals for certification received from the certifying boards and make recommendations concerning each proposal to the Board of Trustees of the American Osteopathic Association for its action.

Sec. h. To maintain contact with the activities of the certifying boards in order to ascertain their conformity to the standards and procedures adopted and to advise them when indicated regarding corrections in their procedures. If necessary, the Advisory Board shall make appropriate recommendations to the Board of Trustees of the American Osteopathic Association when any certifying board persists in failing to conduct its affairs in an acceptable manner.

Sec. i. To cooperate with the Executive Secretary of the American Osteopathic Association in maintaining an accurate listing of certified specialists in good standing.

Sec. j. To assist in educating the profession at large concerning the significance of specialty certification and its relation to the general practitioner.

Sec. k. To respond in an advisory capacity to any group within the profession in matters pertaining to the recognition or declaration of specialization.

Sec. l. To cooperate with the Bureau of Professional Education and Colleges, the Bureau of Hospitals, and the American Osteopathic Association in creating and standardizing specialty training programs and in establishing the principle of certification in the personnel of the organizations coming under its jurisdiction.

Sec. m. To keep a record of all meetings, transactions, and actions of the Advisory Board and maintain in an appropriate form a cumulative compilation of recommendations of this Board to the Board of Trustees of the American Osteopathic Association.

The original stenographic notes of the proceedings of the Advisory Board shall be filed as the legal record of the minutes of the Advisory Board. The edited minutes shall be considered legal after approval by the Advisory Board except in instances of question as to original intent and purpose, when the original record shall become a part of the final document of record.

Sec. n. To establish and maintain a file of the constitutions and bylaws of all approved certifying boards together with their amendments, cumulative lists of their memberships, cumulative lists of those certified by each certifying board, and record the standing of each certificate holder.

ARTICLE III—MEMBERSHIP

Sec. a. The membership of the Advisory Board for Osteopathic Specialists shall consist of those designated ex-officio members together with the secretary-treasurers of the several approved certifying boards and one other appointed representative from each of the certifying boards and representatives from each of the following groups as indicated below, appointed by its chairman or president:

(1) Board of Trustees of the American Osteopathic Association—two representatives.

(2) Bureau of Professional Education and Colleges—two representatives.

(3) Bureau of Hospitals—two representatives.

(4) American Association of Osteopathic Colleges—two representatives.

(5) Central Office—American Osteopathic Association—one representative. The Executive Secretary of the American Osteopathic Association shall appoint one osteopathic physician from Central Office to serve as a member of the Advisory Board.

Sec. b. Member organizations shall make their appointments within 60 days following each annual meeting of the American Osteopathic Association and the appointments shall be communicated promptly to the Executive Secretary of the American Osteopathic Association and to the Secretary of the Advisory Board.

Sec. c. Each member organization is entitled to two votes, but in no case may a member organization be entitled to vote without a duly authorized representative being present. Both votes may be cast by one representative in the absence of the other. The representative of the Central Office shall have no vote.

Sec. d. Alternates may be accepted as members if they are designated by the appropriate member organization officer and their names are in the hands of the Executive Secretary of the American Osteopathic Association and the Secretary of the Advisory Board for Osteopathic Specialists before the time of the annual meeting of the Advisory Board.

Sec. e. When one individual is named by two different member organizations as a representative, the appointing officer whose appointment was last received shall be informed of the previous appointment of the individual by another member organization so that his appointment may be changed if desired.

Sec. f. When an ex officio member of the Advisory Board is appointed as a representative of a member organization, he shall vote in the capacity as such a representative. If he is an Advisory Board member ex officio only, he may vote as an individual.

Sec. g. Any organization which holds membership on the Advisory Board and which does not have its representatives in attendance for two consecutive years shall have its membership on the Advisory Board revoked.

ARTICLE IV—OFFICERS

Sec. a. The officers of this Board shall be Chairman, Vice-Chairman, and Secretary.

Sec. b. The Chairman shall be elected at each alternate annual meeting for a term of 2 years from those who are members at the time of election.

In the event of his not being a member of the Advisory Board under the provisions of Article III, Sec. a, during the period for which he is elected, he shall continue a member for the term of his office.

He shall appoint all committees, excepting as otherwise provided, serve as an ex-officio member of all standing committees, make the prescribed reports, preside at all meetings of the Advisory Board, act as Chairman of the Executive Committee, and perform such other duties as customarily reside in the office.

He shall make an annual and a midyear report of the Advisory Board's activities to the Board of Trustees of the American Osteopathic Association.

Sec. c. The Vice-Chairman shall be elected at each alternate annual meeting for a term of 2 years from those who are members at the time of the election.

He shall be a member of the Advisory Board at the time of his election and shall be a member ex officio during the term for which he is elected.

He shall perform the duties customarily resident in this office.

Sec. d. The Secretary shall be elected every third year for a term of 3 years from those who are members at the time of the election.

He shall be a member of the Advisory Board ex officio during the term for which he is elected.

He shall keep a record of all meetings, transactions, and actions of the Advisory Board and assist the Chairman in such other duties as are appropriate to this office.

Sec. e. In the event of a vacancy in the office of the Chairman and Vice-Chairman, the senior member of the two members at large elected to the Executive Committee, shall act as Chairman until the next regular meeting.

ARTICLE V—COMMITTEES

Sec. a. The Executive Committee shall be composed of the Chairman, Vice-Chairman, Secretary, and two members of the Advisory Board.

One such member from the Advisory Board shall be elected each year for a term of 2 years.

Elected members of the Executive Committee shall be members ex officio of the Advisory Board through the terms for which they are elected.

This committee shall carry out the policy and activity initiated or decided by the Advisory Board. The Executive Committee shall meet on call of the Chairman.

The Executive Committee shall act for the Advisory Board between meetings of the Advisory Board. All actions of the Executive Committee shall be subject to approval of the Board of Trustees of the American Osteopathic Association.

Sec. b. A Review Committee composed of three members shall be appointed by the Chairman not later than 30 days before the time of the annual meeting, or special meeting, from among members who affirm their intent to be in attendance at the meeting. This committee shall review the data supporting the proposals for certification submitted by the certifying boards and make recommendation to the Advisory Board concerning the proper action on each candidate proposed.

Names of the candidates who fail to meet the requirements of the Review Committee shall not be presented to the Advisory Board.

Sec. c. A Nominating Committee composed of three members shall be appointed by the Chairman at the time of the annual meeting to name candidates for officers for the ensuing year and to report to the last session of the meeting, at which time election of officers shall take place. Nominations from the floor will be in order.

Sec. d. An Appeal Committee shall be selected and appointed by the Chairman of the Advisory Board, with the approval of the Executive Committee whenever it is deemed necessary by the Chairman of the Advisory Board to provide for adjudication of a charge filed with the Advisory Board of unfairness or improper conduct of the affairs of any certifying board by an applicant for certification.

The Appeal Committee shall be composed of five members, who shall elect their own chairman, one individual to be appointed from the Bureau of Professional Education and Colleges; one from the Bureau of Hospitals; one from the Board of Trustees of the American Osteopathic Association; and two from the membership of the Advisory Board for Osteopathic Specialists, no one of whom shall be a member of the certifying board which is the object of complaint.

The meeting, or meetings, of the committee shall be arranged by the Chairman of the Appeal Committee with the complainant and a representative appointed by the chairman of the certifying board involved. A stenotype record of the proceedings shall be made, and such notes shall be confidential.

After hearing and considering the presentation of the complaint and the certifying board's response giving reasons for the action against which the complaint is directed, and after making any necessary investigation, the Appeal Committee shall make a report to the Advisory Board for Osteopathic Specialists, indicating what the committee considers to be the proper action to be followed by the parties concerned. If the controversy is not suitably adjudicated and the circumstances justify, the report of the Appeal Committee shall be presented to the Board of Trustees of the American Osteopathic Association for further action.

This committee shall be dissolved upon conclusion of the duties specified above.

Sec. e. A Committee on Basic Documents shall be appointed by the Chairman of the Advisory Board for Osteopathic Specialists at each annual meeting. For the purposes of this Committee, the basic documents shall be defined as the Constitution and Bylaws, Regulations and Requirements of the specialty certifying boards, and the Manual of Procedure for the Advisory Board for Osteopathic Specialists and Boards of Certification.

The duties of this Committee shall be:

(1) To maintain a file of all basic documents of the Advisory Board and its member certifying boards.

(2) To prepare and submit to the Advisory Board amendments to the Manual of Procedure for the Advisory Board for Osteopathic Specialists and Boards of Certification.

(3) To review the basic documents of the specialty certifying boards and recommend proposed changes in these documents as may be necessary to establish conformity with the Manual of Procedure for the Advisory Board for Osteopathic Specialists and Boards of Certification, as approved by the Board of Trustees of the American Osteopathic Association.

ARTICLE VI—MEETINGS

Sec. a. A regular annual meeting shall be held at the time and place of the annual convention of the American Osteopathic Association. The meeting shall be scheduled at such time that recommendations from this Advisory Board may be transmitted to the Board of Trustees of the American Osteopathic Association before the conclusion of its final sessions. The time and place of the sessions of the Advisory Board's meeting shall be communicated to all members not less than 30 days before the date of convening.

Sec. b. A quorum at any session of any regular meeting shall consist of a majority of the designated membership of the Advisory Board, and at least one-third of the member organizations shall be represented.

Sec. c. Robert's Rules of Order shall be followed except where they conflict with these rules of procedure.

Sec. d. The order of business shall be as follows:

(1) Call to order.
(2) Roll call.
(3) Secretary's report including:
  (a) Presentation of the report to the Board of Trustees of the American Osteopathic Association made at the preceding annual meeting.
  (b) Presentation of the Chairman's preliminary report to the Board of Trustees of the American Osteopathic Association, and the recommendations for the current session of the Advisory Board.
(4) Appointment of Nominating Committee.
(5) Reports and recommendations of certifying boards.
(6) Reports of standing committees.
(7) Reports of special committees.
(8) Unfinished business.
(9) Receipt of communications.
(10) Report of Review Committee.
(11) New business and assignments to appropriate committees.
(12) Report of Nominating Committee and election of officers.
(13) Appointment of committees for ensuing year.
(14) Adjournment.

ARTICLE VII—AMENDMENTS

Sec. a. Amendments to these Rules of Organization and Procedure may be adopted for submission to the Board of Trustees of the American Osteopathic Association for approval by the vote of a majority of the members of the Advisory Board registered with the Secretary as present at the annual meeting, provided the proposed amendment has been submitted to each member present in writing or printed form not less than 12 hours preceding the time of the meeting at which the vote is to be taken.

Sec. b. A Manual of Procedure for the Advisory Board for Osteopathic Specialists and Boards of Certification shall be issued by Central Office within 90 days following adoption of this recommendation by the Board of Trustees. It shall contain the following:

(1) Rules of Organization and Procedure of the Advisory Board for Osteopathic Specialists as amended with the approval of the Advisory Board and the Board of Trustees.

(2) Rules of Procedure for specialty certifying boards as amended with the approval of the Advisory Board and Board of Trustees.

(3) Standard Constitution and Bylaws of Boards of Certification as approved by the Board of Trustees.

(4) Model form for Regulations and Requirements of Boards of Certification.

(5) Model form Outline for Annual Report of Secretaries of Certifying Boards to the Advisory Board.

Sec. c. The documents listed under Section b above shall be edited, rearranged, and revised, without changing the intent, to provide better phraseology and continuity. Repetitions shall be deleted.

Sec. d. Three hundred copies of the Manual shall be issued. They shall be distributed to the Executive Committee of the American Osteopathic Association, officers and members of boards of certification, and to other individuals who have a right to the Manual.

Sec. e. The Manual shall be revised every 2 years, and the revisions shall include recommendations of the Advisory Board which have been approved by the Board of Trustees since the preceding issue of the Manual pertaining to the conduct of the Advisory Board, boards of certification, and applicants for certification.

Sec. f. All recommendations which amend or in any way alter any of the documents reproduced in the Manual shall be presented as amendments with proper reference to the document, article, section, and paragraph.

BOARDS OF SPECIALTY CERTIFICATION

STANDARD CONSTITUTION AND BYLAWS

(Approved by the Board of Trustees of the American Osteopathic Association, July, 1948)

CONSTITUTION
of the
AMERICAN OSTEOPATHIC BOARD OF.....

ARTICLE I—NAME

The name of this organization shall be The American Osteopathic Board of .....

ARTICLE II—PURPOSE

Sec. 1. The purposes of this Board shall be:

(a) To define the qualifications to be required of osteopathic physicians for certification in the field (or fields) of ..... and of any other specialty field that may be assigned to this Board.

(b) To determine the qualifications of osteopathic physicians as specialists for certification in the field (or fields) of ..... and of any other specialty field that may be assigned to it.

(c) To conduct examinations in conformity with the Bylaws of this Board.

(d) To issue certificates, subject to the recommendation of the Advisory Board for Osteopathic Specialists and to the approval of the Board of Trustees of the American Osteopathic Association, to those physicians who are found qualified.

(e) To recommend revocation of certificates for cause.

(f) To use every means possible to maintain a high standard of practice in these specialties (this specialty) within the osteopathic profession.

The actions of the American Osteopathic Board of ..... are subject to the recommendation of the Advisory Board for Osteopathic Specialists and to the approval of the Board of Trustees of the American Osteopathic Association.

ARTICLE III—DEFINITION

Sec. 1. For the purpose of the operation of this Board the following division(s) of practice is (are) defined:

(a) The practice of ..... shall consist of and include .....
.....

(b) The practice of ..... etc., etc.

ARTICLE IV—ORGANIZATION

Sec. 1. Membership.

(a) The American Osteopathic Board of ..... shall consist of ..... members elected by .....
.....
Each elected member shall be a certified specialist in good standing in .....
(Each specialty group shall be represented in the membership of the Board .....) Insofar as possible there shall be a representative from each geographical time belt of the United States on this Board.

(b) Members shall be elected for a term of 3 years (except that at the first election of members to this Board three members shall be elected for 3 years, three shall be elected for 2 years, and three for 1 year. Thereafter, three members shall be elected annually for the full term of 3 years.) The terms of members should be so staggered that the new members elected in any year shall not constitute a majority of the Board. In case of death or resignation of any Board member, ... shall appoint a certified specialist from the corresponding field to fill the unexpired portion of the term.

(c) Members shall continue to serve until their successors are elected.

Sec. 2. Officers.

(a) The officers of this Board shall be:

(i) Chairman.
(ii) Vice-Chairman.
(iii) Secretary-Treasurer.

(b) These officers shall be elected by this Board for a term of 1 year at its annual meeting.

(c) Officers shall continue to hold office until their successors are elected.

Sec. 3. Committees.

(a) There shall be three standing committees of this Board and such other committees as may from time to time be authorized. The Chairman shall appoint all committees unless it is otherwise provided.

(b) The standing committees shall be:

(i) Credentials Committee.
(ii) Examination Committee.
(iii) Advisory Board Committee.

ARTICLE V—AMENDMENT

Sec. 1. Subject to the review and recommendation of the Advisory Board for Osteopathic Specialists and the approval of the Board of Trustees of the American Osteopathic Association this Constitution may be amended by a vote of two thirds of the total membership of this Board (or three fifths in a five-member Board) at any annual meeting following notification given at any previous annual meeting of such intention.

BYLAWS
of the
AMERICAN OSTEOPATHIC BOARD OF.....

ARTICLE I
DUTIES OF THE AMERICAN OSTEOPATHIC BOARD OF .....

Sec. 1. This Board shall serve as an advisory body to all applicants for certification in the specialty (specialties) of ..... and any other specialty which may be assigned to its jurisdiction.

Sec. 2. This Board shall determine, in accordance with the provisions of these Bylaws, the appropriate standards of education and the training held necessary for certification in the specialty (specialties) of ..... and of any other specialty which may be assigned to its jurisdiction, subject to the recommendations of the Advisory Board for Osteopathic Specialists and to the approval of the Board of Trustees of the American Osteopathic Association.

Sec. 3. This Board shall establish detailed rules for conducting all examinations in compliance with the provisions of these Bylaws and shall provide for the conduct of examinations at least once a year in accordance with its Regulations and Requirements.

Sec. 4. This Board shall file with the Advisory Board for Osteopathic Specialists, at the time specified by the Advisory Board, its recommendations concerning each applicant for certification together with any pertinent information required by the Advisory Board for Osteopathic Specialists.

Sec. 5. This Board shall provide and issue certificates in all fields assigned to this Board's jurisdiction, as provided in Article V of these Bylaws.

Sec. 6. This Board shall provide permanent files for all records. It shall record and keep permanently on file all applications submitted to it and complete records of examination

results and shall maintain a registry of certificate holders. All examination papers and case reports of applicants shall be kept on file for a period of 3 years.

Sec. 7. This Board shall levy and collect from applicants the funds necessary to finance the operation of this Board as provided in the Regulations and Requirements.

Sec. 8. This Board shall arrange for all meetings necessary to enable this Board to carry out its functions as provided in Article VI, Sections 1 and 2 of these Bylaws.

Sec. 9. This Board shall provide two members of this Board to function as representatives to the Advisory Board for Osteopathic Specialists. One of the two members shall be the Secretary-Treasurer. In case of inability of the regular representatives to attend the sessions of the Advisory Board for Osteopathic Specialists, the Chairman of this Board shall appoint alternates as provided in the Rules of Organization and Procedure of the Advisory Board for Osteopathic Specialists.

Sec. 10. This Board shall conduct its activities in relation to the officers of the American Osteopathic Association, the Advisory Board for Osteopathic Specialists, other certifying boards, and applicants for certification as provided in the Rules of Procedure for Specialty Certifying Boards compiled by the Advisory Board for Osteopathic Specialists and approved by the Board of Trustees of the American Osteopathic Association.

Sec. 11. This Board shall make, in conformity to its Constitution and Bylaws, all necessary regulations and requirements to govern its activities which are not provided by the Advisory Board for Osteopathic Specialists and the Board of Trustees of the American Osteopathic Association.

ARTICLE II—COMMITTEES

Sec. 1. Credentials Committee.

(a) There shall be a Credentials Committee of ..... members appointed by the Chairman of this Board, whose members shall represent different geographical districts so far as possible.

(b) It shall be the duty of the Credentials Committee:

(i) to receive all completed applications from the Secretary-Treasurer;

(ii) to conduct a comprehensive investigation of each applicant in accordance with the rules governing application;

(iii) to prepare a recommendation concerning each applicant and present this to the American Osteopathic Board of ..... at its next (annual) meeting.

Sec. 2. Examination Committee.

(a) There shall be an Examination Committee composed of the Chairman of this Board and not less than two additional Board members. The Chairman of this Board shall act as the Chairman of the Examination Committee.

(b) It shall be the duty of the Examination Committee to plan and to make preparations for the conduct of examinations in the special fields coming under the jurisdiction of this Board in accordance with the rules governing examinations as stated in Article IV, Section 1, Paragraph (h) of these Bylaws and in the Regulations and Requirements of this Board.

(c) The Examination Committee shall report to this Board the findings on the examination of each applicant.

Sec. 3. Advisory Board Committee.

(a) There shall be an Advisory Board Committee consisting of the Chairman and the Secretary-Treasurer of this Board.

(b) It shall be the duty of the Advisory Board Committee to represent the American Osteopathic Board of .... in all matters where such representation is required.

(c) The Chairman shall annually appoint as an alternate member of this Committee a member of the Board of .... who shall be empowered to act for either regular member who is not in attendance.

ARTICLE III—DUTIES OF OFFICERS

Sec. 1. The duties of the Chairman shall be:

(a) to preside at all meetings;

(b) to appoint all committees and representatives to the Advisory Board for Osteopathic Specialists;

(c) to schedule meetings of this Board at such times and places as necessary to carry on the business of this Board;

(d) to supervise all examinations;

(e) to act as an ex officio member of all committees;

(f) to sign all certificates issued by this Board;

(g) to act as Chairman of the Examination Committee;

(h) to be a member of the Advisory Board Committee.

Sec. 2. The duties of the Vice-Chairman shall be:

(a) to assume the duties of the Chairman when the latter is absent or otherwise unable to fulfill them;

(b) to assist the Chairman in the discharge of his duties.

Sec. 3. The duties of the Secretary-Treasurer shall be:

(a) to keep in permanent file records of all proceedings, transactions, and rulings of this Board, and to keep on file all examination papers and case records for a period of 3 years;

(b) to have printed and distributed all certificates, application forms, circulars of information, etc., authorized by this Board and necessary for the proper functioning of this Board;

(c) to maintain in proper place and form the moneys of this Board and to issue an accounting of them at annual meetings or at such other times as requested by the Chairman of this Board;

(d) to keep a permanent file of all applications coming before this Board with a signed record of the action taken on each;

(e) to maintain a record of all certificate holders in good standing and to supply upon request to governmental agencies, hospitals, physicians, schools, and others entitled to such information, a list of holders of certificates in good standing;

(f) to have prepared in appropriate and complete form for presentation to the Review Committee of the Advisory Board for Osteopathic Specialists in support of recommendations for certification the application, examination record (written, oral, and practical), and other pertinent information requested; (See Rules of Procedure.)

(g) to serve as a representative of this Board to the Advisory Board for Osteopathic Specialists;

(h) to sign all certificates issued by this Board, as provided in Article V;

(i) to prepare an annual report in keeping with the Rules of Procedure for Specialty Certifying Boards of the work done by this Board, including a list of all applicants and the results of their examinations and a resume of the Board's finances. This report is to be presented at the annual meeting of the Advisory Board for Osteopathic Specialists and copies are to be supplied to the Chairman and Secretary of the Advisory Board and to the Executive Secretary of the American Osteopathic Association;

(j) to cooperate with the Executive Secretary of the American Osteopathic Association in all matters pertaining to the annual registration of certificate holders;

(k) to notify the Executive Secretary of the American Osteopathic Association and the Chairman and Secretary of the Advisory Board for Osteopathic Specialists of members and officers elected to this Board and of appointments to the Advisory Board for Osteopathic Specialists.

ARTICLE IV—REQUIREMENTS FOR CERTIFICATION

Sec. 1. To be eligible to receive a certificate of specialization from the American Osteopathic Board of ..... the applicant must meet all of the following minimum requirements, excepting as provided in Section 3 (and Section 4) of this Article:

(a) He must be a graduate of an approved osteopathic college.

(b) He must be licensed to practice in the state or territory where he conducts his specialty practice.

(c) He must have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years immediately prior to application for examination by this Board.

(d) He must have satisfactorily completed an internship of at least 1 year in a hospital approved for intern training by the American Osteopathic Association or satisfactory to this Board. If graduated in 1942 or prior thereto, the applicant must have had training of value equivalent to that of an acceptable internship as determined by this Board.

(e) He must be able to show evidence of conformity to the standards set in the Code of Ethics of the American Osteopathic Association.

(f) A period of not less than 3 years of special training related to the specialty shall be required, after the required 1 year of internship, or its equivalent, but not necessarily running 3 consecutive years. This Board may modify the requirement of 3 years' special training by allowing a credit of 1 year of special training for each 5 years of specialty practice for a total credit of not to exceed 2 years of the requirement, for physicians who graduated prior to 1946, but in no case may such applicant be accepted for examination without at least 1 year of special training as defined. This training shall include:

(i) Advanced study in the sciences: Anatomy, physiology, biochemistry, pathology, pharmacology, and such other basic sciences as are necessary to a mature and comprehensive understanding of the prevention, diagnosis, therapy, and management of the disorders involved in the specialty.

(ii) Active experience in the diagnosis and treatment in such amount and diversity that it will assure adequate training in the specialty.

The special training may be provided in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board, or by serving an acceptable assistantship to or studying under the preceptorship of a qualified specialist in this field, supplemented by prescribed study conforming to a plan approved by this Board.

The minimum 3-year period of special training may be made up of varying combinations of the several types mentioned above, as determined by this Board. It shall be this Board's duty to assist applicants in determining a suitable program of special training, but it is not this Board's function to supply the training. This Board will recognize as proper institutions for securing this training those institutions which are approved for special training by the approving agencies of the American Osteopathic Association, together with such others as this Board may determine to possess facilities and personnel adequate to provide proper training.

(g) He must practice as a specialist in his field for a period of at least 2 years subsequent to the minimum 3 years of special training. Specialty practice within each specialty field under this Board shall be defined in the Regulations and Requirements of this Board.

(h) Following satisfactory compliance with the prescribed requirements as to special training and specialty practice, the applicant shall be required to pass appropriate examinations planned to evaluate his understanding of the scientific bases of the problems involved in the specialty, his familiarity with the current advances in the specialty, and the possession of sound judgment and of a high degree of skill in the diagnostic and therapeutic procedures involved in the practice of the specialty. Oral, written, and clinical examinations shall be conducted and required in the case of each applicant. This Board may conduct the oral and written examinations at the end of the required 3 years of special training, but shall not conduct the practical or clinical examination until the required 2 years of specialty practice have been completed. The members of this Board shall personally review, if not perform, the grading of each written examination. The conduct of the clinical examinations may be delegated to committees of not fewer than two individuals maturely qualified in the specialty. A full description of the method of conducting the examination shall be formulated in this Board's Regulations and Requirements, and provision for re-examination shall be made.

(i) Applicants desiring examination for certification shall be required to file an application which shall set forth the applicant's qualifications for examination as stated in paragraphs (a), (b), (c), (d), (e), (f), and (g) above. The procedure for filing applications shall be set forth in the Regulations and Requirements and no application for action at any annual meeting of this Board shall be accepted after April 1st of that year.

Sec. 2. In the case of any applicant who was engaged in the practice of his specialty by 1939, or prior thereto, and who graduated prior to 1936, and who meets the general requirements stated in Section 1, (a), (b), (c), (d), and (e) above, and has maintained appropriate activity in American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgraduate work, contributions to professional literature or otherwise has indicated his consistent interest and competence in his specialty, the requirements of this Article IV, Section 1, paragraphs (f) and (h) may be waived and he may be recommended for certification after passing an appropriate oral and/or clinical examination.

Sec. 3. Subject to the recommendations of the Advisory Board for Osteopathic Specialists and to the approval of the Board of Trustees of the American Osteopathic Association, the American Osteopathic Board of ..... may require such further training in each of the specialty fields coming under its jurisdiction as in its judgment such field may require, provided that the additional requirements for each specialty field are clearly set forth in the Regulations and Requirements of this Board. Additions to requirements shall not go into effect until 1 year subsequent to the announcement of such change.

Sec. 4. If circumstances necessitate it, a section may be written and inserted at this point to provide for the fair handling of applicants already acting on the basis of commitments made by this Board prior to 12-15-46.

ARTICLE V—CERTIFICATES

Sec. 1. Issuance.

Certificates of specialization which have been approved by the Board of Trustees of the American Osteopathic Association shall be issued by the American Osteopathic Board of ..... to applicants who have conformed to all requirements for certification described in Article IV of these Bylaws and who have received the recommendation of the Advisory Board for Osteopathic Specialists and the approval of the Board of Trustees of the American Osteopathic Association. Each such certificate shall be signed by the Chairman and the Secretary-Treasurer of this Board. No certificate is valid until it has been signed by the Executive Secretary of the American Osteopathic Association.

Sec. 2. No certificate shall be issued to an individual who is certified by another board, until the former certificate has been surrendered to the Executive Secretary of the American Osteopathic Association.

Sec. 3. Revocation.

This Board shall have the power to recommend to the Advisory Board for Osteopathic Specialists and the Board of Trustees of the American Osteopathic Association the revocation of the certificate of any holder whose certificate was obtained by fraud or misrepresentation, who unduly advertises, exploits his certificate, violates the Code of Ethics of the American Osteopathic Association, or otherwise disqualifies himself.

Sec. 4. To remain in good standing the certificate holder shall pay an annual registration fee to the Executive Secretary of the American Osteopathic Association, as provided in the Rules of Procedure for Specialty Certifying Boards, as compiled by the Advisory Board for Osteopathic Specialists.

ARTICLE VI—MEETINGS

Sec. 1. The American Osteopathic Board of ..... shall hold a regular annual meeting to transact business and to conduct examinations at the time and place of the annual meeting of the American Osteopathic Association (or at the annual meeting of its respective specialty college).

Sec. 2. Special meetings of this Board which are deemed necessary for the transaction of business may be called with the approval of the majority of the members of this Board provided all members are formally notified of the called meet-

ing and its purpose, not less than 30 days before the date set for the meeting.

Sec. 3. For the transaction of business at any meeting of this Board, ... members shall constitute a quorum.

ARTICLE VII—AMENDMENT

Sec. 1. Subject to the review and recommendation of the Advisory Board for Osteopathic Specialists and to the approval of the Board of Trustees of the American Osteopathic Association, these Bylaws may be amended by a two-thirds vote of the total membership of this Board (or three-fifths in a five-member Board) at any meeting provided each member has been notified at least 30 days prior to the date of the meeting of its being called and of the intention to amend.

AMERICAN OSTEOPATHIC BOARD OF DERMATOLOGY AND SYPHILOLOGY

Ronald W. MacCorkell, Chairman, Los Angeles
Edwin H. Cressman, Vice Chairman, Philadelphia
Anthony E. Scardino, Kansas City, Mo.
Cecil D. Underwood, Los Angeles
James D. Stover, Secretary-Treasurer, Detroit

DEFINITION OF SPECIALTY PRACTICE

For the purpose of the operation of this Board the following divisions of practice are defined:

a. The practice of dermatology shall consist of and include the diagnosis and the surgical and nonsurgical management of the diseases of the skin and its appendages.

b. The practice of syphilology shall consist of and include the diagnosis and the surgical and nonsurgical management of syphilis in all its phases and stages.

c. A specialty practice, as defined by this Board, shall consist of not less than 90 per cent dermatology and syphilology.

REQUIREMENTS FOR CERTIFICATION

Section 1. To be eligible to receive a certificate of specialization from the American Osteopathic Board of Dermatology and Syphilology, the applicant must meet all of the following minimum requirements, excepting as provided in Section 2 of this Article:

(a) He must be a graduate of an approved osteopathic college.

(b) He must be licensed to practice in the state or territory where he conducts his specialty practice.

(c) He must have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years immediately prior to application for examination by this Board.

(d) He must have satisfactorily completed an internship of at least 1 year in a hospital approved for intern training by the American Osteopathic Association or satisfactory to this Board. If graduated in 1942 or prior thereto, the applicant must have had training of value equivalent to that of an acceptable internship as determined by this Board.

(e) He must be able to show evidence of conformity to the standards set in the Code of Ethics of the American Osteopathic Association.

(f) A period of not less than 3 years of special training related to the specialty shall be required, after the required 1 year of internship, or its equivalent, but not necessarily running 3 consecutive years. This Board may modify the requirement of 3 years' special training by allowing a credit of 1 year of special training for each 5 years of specialty practice for a total credit of not to exceed 2 years of the requirement, for physicians who graduated prior to 1946, but in no case may such applicant be accepted for examination without at least 1 year of special training as defined. This training shall include:

(i) Advanced study in the sciences: Anatomy, physiology, biochemistry, pathology, pharmacology, and such other basic sciences as are necessary to a mature and comprehensive understanding of the prevention, diagnosis, therapy, and management of the disorders involved in the specialty.

(ii) Active experience in the diagnosis and treatment in such amount and diversity that it will assure adequate training in the specialty.

The special training may be provided in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board, or by serving an acceptable assistantship to, or studying under the preceptorship of, a qualified specialist in this field, supplemented by prescribed study conforming to a plan approved by this Board.

The minimum 3-year period of special training may be made up of varying combinations of the several types mentioned above, as determined by this Board. It shall be this Board's duty to assist applicants in determining a suitable program of special training, but it is not this Board's function to supply the training. This Board will recognize as proper institutions for securing this training those institutions which are approved for special training by the approving agencies of the American Osteopathic Association, together with such others as this Board may determine to possess facilities and personnel adequate to provide proper training.

(g) He must practice as a specialist in his field for a period of at least 2 years subsequent to the minimum 3 years of special training. Specialty practice within each specialty field under this Board shall be defined in the Regulations and Requirements of this Board.

(h) Following satisfactory compliance with the prescribed requirements as to special training and specialty practice, the applicant shall be required to pass appropriate examinations planned to evaluate his understanding of the scientific bases of the problems involved in the specialty, his familiarity with the current advances in the specialty, the possession of sound judgment and of a high degree of skill in the diagnostic and therapeutic procedures involved in the practice of the specialty. Oral, written, and clinical examinations shall be conducted and required in the case of each applicant. This Board may conduct the oral and written examinations at the end of the required 3 years of special training, but shall not conduct the practical or clinical examination until the required 2 years of specialty practice have been completed. The members of this Board shall personally review, if not perform, the grading of each written examination. The conduct of the clinical examinations may be delegated to committees of not fewer than two individuals maturely qualified in the specialty. A full description of the method of conducting the examination shall be formulated in this Board's Regulations and Requirements, and provision for re-examination shall be made.

(i) Applicants desiring examination for certification shall be required to file an application which shall set forth the applicant's qualifications for examination as stated in paragraphs (a), (b), (c), (d), (e), (f), and (g) above. The procedure for filing applications shall be set forth in the Regulations and Requirements and no application for action at any annual meeting of the Board shall be accepted after April 1st of that year.

Section 2. In the case of any applicant who was engaged in the practice of his specialty by 1939 or prior thereto, and who graduated prior to 1936 and who meets the general requirements stated in Section 1, (a), (b), (c), (d), and (e) above and has maintained appropriate activity in American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgraduate work, contributions to professional literature or otherwise has indicated his consistent interest and competence in his specialty, the requirements of this Article IV, Section 1, paragraphs (f) and (h) may be waived, and he may be recommended for certification after passing an appropriate oral and/or clinical examination.

Section 3. Subject to the recommendations of the Advisory Board for Osteopathic Specialists and to the approval of the Board of Trustees of the American Osteopathic Association, the American Osteopathic Board of Dermatology and Syphilology may require such further training in each of the specialty fields coming under its jurisdiction as in its judgment such field may require, provided that the additional requirements for each specialty field are clearly set forth in the Regulations and Requirements of this Board. Additions to requirements shall not go into effect until 1 year subsequent to the announcement of such change.

AMERICAN OSTEOPATHIC BOARD OF INTERNAL MEDICINE

H. Earle Beasley, Chairman, Boston
William F. Daiber, Vice Chairman, Philadelphia
Maxwell R. Brothers, Los Angeles
John L. Crowther, Bangor, Me.
Ralph E. Everal, Detroit
Stuart F. Harkness, Des Moines, Iowa
Basil Harris, Los Angeles
Neil R. Kitchen, Detroit
Glennard E. Lahrson, Secretary-Treasurer, Oakland, Calif.

DEFINITION OF SPECIALTY PRACTICE

The practice of internal medicine is devoted primarily to the application of subjective, objective, and laboratory procedures in studying and evaluating the affections, diseases, and functional disorders of the circulatory, respiratory, digestive, endocrine, metabolic, musculoskeletal, hematopoietic, and eliminative systems of the human body to:

a. the correlation of data thus secured with clinical observations to reach diagnostic conclusions;
b. the management of cases within this field by the utilization of all nonsurgical measures;
c. assist in planning the therapeutic utilization of the technics of other specialty branches of medical and surgical practice in the care of patients.

The practice of internal medicine does not include the treatment of the organs of special sense or the organs of the reproductive system, except as to their endocrine relations, and excludes the use of instrumental procedures ordinarily performed by surgeons or surgical specialists.

REQUIREMENTS FOR CERTIFICATION

A. *Educational Qualifications.*—To be eligible to receive a certificate of specialization from the American Osteopathic Board of Internal Medicine the applicant must meet all of the minimum requirements as set forth in the Bylaws of the American Osteopathic Board of Internal Medicine, Article IV, Sections 1, 2, and 3.

The 3-year period of training related to the specialty as required in the Bylaws, Article IV, Section 1, (f) is further defined:

1. A period of not less than 3 years of special training related to this specialty shall be required, after the required 1 year of internship or its equivalent, but not necessarily running 3 consecutive years. This Board may modify the requirement of 3 years' special training by allowing a credit of 1 year of special training for each 5 years of specialty practice for a total credit of not to exceed 2 years of the requirement for physicians who graduated prior to 1946, but in no case may such applicant be accepted for examination without at least 1 year of special training as defined.

A year of special training shall be interpreted to mean 12 months; a training hour is interpreted to mean a platform hour. This training shall include:

1. Advanced study in the sciences: Anatomy, physiology, biochemistry, pathology, pharmacology, and such other basic sciences as are necessary to a mature and comprehensive understanding of the prevention, diagnosis, therapy, and management of the disorders involved in the specialty.

2. Active experience in the diagnosis and treatment in such amount and diversity that it will assure adequate training in the specialty.

3. To obtain a mature and comprehensive understanding of the Basic Sciences this Board recommends a minimum of 160 hours of basic science instruction on the graduate level, divided as follows:

Anatomy	30 hours
Physiology	37 hours
Pathology	37 hours
Biochemistry	15 hours
Bacteriology	20 hours
Pharmacology	21 hours

4. Didactic and clinical instruction to assure adequate training in this specialty is recommended in the various fields of internal medicine totaling 320 hours. This instruction must include cardiology, electrocardiography, peripheral vascular diseases, medical diseases of the kidney, gastroenterology, metabolic diseases, endocrinology, hematology, clinical pathology, respiratory diseases, diseases of the biliary system, musculoskeletal system, x-ray interpretation, ophthalmoscopy, and osteopathic principles and therapeutics including materia medica as these relate to internal medicine.

5. The special training may be provided in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board, which may include:

a. *Preceptorship.*—All preceptorship training programs must be approved by the Committee on Accreditation of Postgraduate Training and the Board of Trustees of the American Osteopathic Association on recommendation of this Board. The preceptorship training program shall be full time for a period of 3 years, but not necessarily running 3 consecutive years. The preceptorship training program shall be previously submitted to and approved by the Committee on Accreditation of Postgraduate Training of the American Osteopathic Association and the Board of Trustees of the American Osteopathic Association on recommendation of this Board. The preceptor and trainee must likewise be qualified and approved by the above agencies. The trainee shall not maintain an independent practice.

b. *Full-time Assistantship.*—The assistantship must extend for a minimum period of 3 years to a certified internist acceptable to this Board. A full explanation of the scope and diversity of practice, together with any other information that may be from time to time requested, shall be presented to this Board for approval.

c. *Residency.*—Full-time residency in internal medicine shall be in a hospital approved for resident training by the Bureau of Hospitals of the American Osteopathic Association. Appropriate credit toward the special training requirement of this Board shall be allowed for full-time approved residencies of 1, 2, or 3 years in internal medicine.

6. It shall be this Board's duty to assist applicants in determining a suitable program of special training, but it is not this Board's function to supply the training. This Board will recognize as proper institutions for securing this training those institutions which are approved for special training by the approving agencies of the American Osteopathic Association, together with such others as this Board may determine to possess facilities and personnel adequate to provide proper training. An affidavit shall be furnished upon the request of this Board.

B. *Membership Requirements.*—Membership qualifications are those as stated in the Bylaws of the American Osteopathic Board of Internal Medicine, Article IV, Section 1, (c).

C. *Graduation Requirements.*—The graduation requirements with exceptions are those as stated in the Bylaws of the American Osteopathic Board of Internal Medicine, Article IV, Section 2.

D. *Years of Specialty Practice Required.*—The applicant must have practiced as a specialist in the field of internal medicine for a period of at least 2 years subsequent to the minimum of 3 years of special training, as defined by this Board, before he is eligible for clinical examination.

E. *Percentage of Practice in Internal Medicine.*—The applicant is expected to have practiced according to the "Definition of the Practice of Internal Medicine" as printed in the Regulations and Requirements of the American Osteopathic Board of Internal Medicine, Part II, 1. An affidavit shall be furnished on request of the Board.

F. *Case Records.*—Specially prepared case records for presentation to this Board are not required, however, during the investigation by the Credentials Committee the applicant's office and/or hospital records shall be made available on request of the Committee. Such original records are not to be removed from the office or hospital.

G. *Copy of Published Articles.*—Authenticated copies of published articles are to be furnished on request of the Credentials Committee. No thesis is required.

H. *Privilege of Re-Examination.*—No applicant will be re-examined within a period of 1 year following an initial failure. Re-application will be necessary to retake the examination. If the applicant fails the second examination, a 2-year period of further special study and practice satisfactory to the American Osteopathic Board of Internal Medicine is required. A re-application will be necessary to retake the examination after the second failure.

AMERICAN OSTEOPATHIC BOARD OF NEUROLOGY AND PSYCHIATRY

K. Grosvenor Bailey, Chairman, Los Angeles
Floyd E. Dunn, Vice Chairman, Kansas City, Mo.
Cecil Harris, Philadelphia
Fred M. Still, Macon, Mo.
Thomas J. Meyers, Secretary-Treasurer, Pasadena, Calif.

DEFINITION OF SPECIALTY PRACTICE

For the purpose of the operation of this Board, the following divisions of practice are defined:

a. The specialty of neurology shall consist of that division of the practice of osteopathic medicine embracing disorders of the organic nervous system. It shall include all diagnostic procedures and examination technics giving an understanding of the problems involved. Included will be conditions affecting the peripheral nerves, as well as the brain and central nervous system, the autonomic nervous system, and endocrine syndromes, and specific conditions such as epilepsy, migraine, tics, etc., are included. It does not include neurosurgery.

b. The specialty of psychiatry includes that division of the practice of osteopathic medicine involving functional and organic disturbances of the mind, emotions, and nervous system. It includes personality problems, addictions, behavior difficulties, insanities, marital problems, and sexual and social anomalies. It covers counseling and psychotherapy in all its aspects, as well as diagnostic methods leading to an understanding of the dynamics of the mind. It includes legal and child psychiatry and the field of psychosomatics. It includes electroshock, hydrotherapy, electronarcosis, and other physical modalities applied to the treatment of the mentally ill. It does not include neurosurgery.

c. The specialty of neurology and psychiatry includes all of the divisions of osteopathic medicine as combined with the definitions of the specialty of neurology and the specialty of psychiatry, as stated in a. and b. above.

Note: For the definition of neurosurgery and for the requirements, see American Osteopathic Board of Surgery.

REQUIREMENTS FOR CERTIFICATION

Sec. 1. To be eligible to receive a certificate of specialization from the American Osteopathic Board of Neurology and Psychiatry the applicant must meet all of the following minimum requirements, excepting as provided in Article IV, Section 3 and Section 4 of the Bylaws:

(a) He must have graduated from an approved osteopathic college.

(b) He must be licensed to practice in the state or territory where he conducts his specialty practice.

(c) He must have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years immediately prior to application for examination by this Board.

(d) He must have satisfactorily completed an internship of at least 1 year in a hospital approved for intern training by the American Osteopathic Association or satisfactory to this Board. If graduated in 1942 or prior thereto, the applicant must have had training of value equivalent to that of an acceptable internship as determined by this Board.

(e) He must be able to show evidence of conformity to the standards set in the Code of Ethics of the American Osteopathic Association.

(f) A period of not less than 3 years of special training related to the specialty shall be required, after the required 1 year of internship, or its equivalent, but not necessarily running 3 consecutive years. This Board may modify the requirement of 3 years' special training by allowing a credit of 1 year of special training for each 5 years of specialty practice for a total credit of not to exceed 2 years of the requirement, for physicians who graduated prior to 1946, but in no case may such applicant be accepted for examination without at least 1 year of special training as defined. For certification in neurology and psychiatry, the training period shall be not less than 5 years. This training shall include:

(i) Advanced study in the sciences; Anatomy, physiology, biochemistry, pathology, pharmacology, and such other basic sciences as are necessary to a mature and comprehensive understanding of the prevention, diagnosis, therapy, and management of the disorders involved in the specialty.

(ii) Active experience in the diagnosis and treatment in such amount and diversity that it will assure adequate training in the specialty.

The 3-year period of special training as required in Section 1, (f), above, is further defined:

1. For certification in Neurology the following shall be included in an acceptable training program, residency, or preceptorship:

The amount of time of training shall total 3 calendar years or 6,000 clock hours and shall include:

Basic Science	100 clock hours
Psychiatry and Psychology	500 clock hours
Neurological Surgery Observation	500 clock hours
Didactic Courses	500 clock hours

A maximum of 1,000 hours may be granted for case reports, 2 hours being allowed for each. A maximum of 500 such reports may be presented. Such reports may be considered if they are submitted in an approved form and signed by the preceptor. Each report must include sufficient information to adequately describe the problem, support of diagnosis, data for differential diagnostic study, an estimate of the prognosis, and a suggested plan of treatment. Clinical training shall include: Electroencephalography, electromyography, reactions of degeneration, neuro-ophthalmoscopy, spinal taps, cisternal puncture, encephalography, ventriculography, stellate ganglion blocks, angiography, myelography, and autopsy studies. The amount of clinical training shall be 3,400 clock hours. Furthermore, the clinical material utilized must include a sampling of: Degenerative diseases, traumatic conditions, convulsive disorders, infective and toxic problems, neoplasms of the nervous system, deficiency states, cerebro-vascular problems, and geriatric and pediatric neurology.

2. For certification in Psychiatry the following shall be included in an acceptable training program, residency, or preceptorship:

The amount of time of training shall total 3 calendar years or 6,000 clock hours and shall include:

Psychological Subjects, including Testing	500 clock hours
Neurology and Neurological Subjects	500 clock hours
Basic Science	100 clock hours

A maximum of 1,000 hours may be granted for case reports, 2 hours being allowed for each. A maximum of 500 such reports may be presented. Such reports may be considered if they are submitted in an approved form and signed by the preceptor. Psychoanalysis to which the applicant has submitted himself for treatment or for training will be credited for a maximum amount of 200 clock hours. The training program for certification in Psychiatry shall be primarily of a clinical nature and must be conducted as actual contact with patients under supervision. The full range of psychiatric conditions shall include: Functional psychotics, neuroses of all kinds, sexual aberrations, organic mental conditions, geriatric psychiatry, pediatric psychiatry, marital problems, neurologic psychiatry, psychosomatics, mental deficiency, epilepsy, narcolepsy, alcoholism, psychopathic personalities, syphilis of the nervous system, and legal psychiatry. In addition, the applicant shall acquire familiarity with the theory of Psychiatry and the technics of: Electro-shock treatment, physiotherapy in psychiatry, hydrotherapy in psychiatry, pharmacology in psychiatry, dietetics in psychiatry, occupational therapy, psychotherapy (at least an operational ability), hypnosis and suggestion, child guidance, marital counseling, elementary psychological testing, and psychiatric examination procedure. Only work done in clinically oriented disciplines will be credited. The policy of this Board is to scrutinize critically all preceptor training programs in psychiatry because of its limitations and its questionable value. Unapproved preceptor training programs will not be recognized by this Board. The minimum amount of clinical training shall be 3,400 clock hours.

3. For certification in Neurology and Psychiatry an acceptable training program shall consist of a minimum of 5 years' full-time training or a total of 10,000 clock hours. This

training shall consist of 2 years devoted exclusively to the study of Neurology and 2 years devoted exclusively to the study of Psychiatry and 1 year of study which shall include material from both fields and the Basic Sciences.

A holder of a certificate in Neurology or a holder of a certificate in Psychiatry shall be required to complete a minimum of 2 years' additional training to qualify for a certificate in Neurology and Psychiatry. The required 2 years' additional training program must be approved by the American Osteopathic Board of Neurology and Psychiatry.

The special training may be provided in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board, or by serving an acceptable assistantship to or studying under the preceptorship of a qualified specialist in this field, supplemented by prescribed study conforming to a plan approved by this Board.

The minimum 3-year period of special training may be made up of varying combinations of the several types mentioned above, as determined by this Board. It shall be this Board's duty to assist applicants in determining a suitable program of special training, but it is not this Board's function to supply the training. This Board will recognize as proper institutions for securing this training those institutions which are approved for special training by the approving agencies of the American Osteopathic Association, together with such others as this Board may determine to possess facilities and personnel adequate to provide proper training.

(g) He must practice as a specialist in his field for a period of at least 2 years subsequent to the minimum 3 years of special training. At least 80 per cent of the applicant's practice must be in the specialty field.

(h) Following satisfactory compliance with the prescribed requirements as to special training and specialty practice, the applicant shall be required to pass appropriate examinations planned to evaluate his understanding of the scientific bases of the problems involved in the specialty, his familiarity with the current advances in the specialty and the possession of sound judgment and of a high degree of skill in the diagnostic and therapeutic procedures involved in the practice of the specialty. Oral, written, and clinical examinations shall be conducted and required in the case of each applicant. This Board may conduct the oral and written examinations at the end of the required 3 years of special training, but shall not conduct the practical or clinical examination until the required 2 years of specialty practice have been completed. The members of this Board shall personally review, if not perform, the grading of each written examination. The conduct of the clinical examinations may be delegated to committees of not fewer than two individuals maturely qualified in the specialty. A full description of the method of conducting the examination shall be formulated in this Board's Regulations and Requirements, and provision for re-examination shall be made.

Sec. 2. In the case of any applicant who was engaged in the practice of his specialty by 1939, or prior thereto, and who graduated prior to 1936, and who meets the general requirements stated in Section 1, (a), (b), (c), (d), and (e) above and has maintained appropriate activity in American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgraduate work, contributions to professional literature, or otherwise has indicated his consistent interest and competence in his specialty, the requirements of Article IV, Section 1, paragraphs (f) and (h) may be waived, and he may be recommended for certification after passing an appropriate oral and/or clinical examination.

Sec. 3. Subject to the recommendations of the Advisory Board for Osteopathic Specialists and to the approval of the Board of Trustees of the American Osteopathic Association, the American Osteopathic Board of Neurology and Psychiatry may require such further training in each of the specialty fields coming under its jurisdiction as in its judgment such field may require, provided that the additional requirements for each specialty field are clearly set forth in the Regulations and Requirements of this Board. Additions to requirements shall not go into effect until 1 year subsequent to the announcement of such change.

AMERICAN OSTEOPATHIC BOARD OF OBSTETRICS AND GYNECOLOGY

Dorothy J. Marsh, Chairman, Los Angeles
A. J. Still, Vice Chairman, Flint, Mich.
Margaret Jones, Kansas City, Mo.
Kenneth A. Scott, Providence, R. I.
Charles J. Mount, III, Los Angeles
Homer R. Sprague, Lakewood, Ohio
Arthur B. Funnell, Denver
Harold C. Bruckner, Flint, Mich.
Jacquelin Bryson, Secretary-Treasurer, Denver

DEFINITION OF SPECIALTY PRACTICE

a. Obstetrics: The practice of obstetrics shall be defined as the diagnosis and management of all states presented in cases of pregnancy from the time of conception through the postpartum period by use of any or all means excepting those involving laparotomy or vaginal cesarian section.

b. Gynecology: The practice of gynecology shall be defined as the diagnosis and treatment of diseases peculiar to the female, and because of the close relationship does not exclude affections of the mammary gland and urinary tract in the female, but does exclude all major surgical procedures.

c. Obstetrical-Gynecological Surgery: The practice of obstetrical-gynecological surgery shall consist of and include that branch of medical science, art, and practice which is concerned with major operative procedures:

(1) Within the abdomen and pelvis upon the female generative system or structures related thereto;

(2) Major plastic procedures upon the vaginal tract by manual and instrumental means;

(3) And mammary gland.

REQUIREMENTS FOR CERTIFICATION

To be eligible to receive a certificate of specialization from the American Osteopathic Board of Obstetrics and Gynecology the applicant must meet all of the following minimum requirements:

a. He must be a graduate of an approved osteopathic college.

b. He must be licensed to practice in the state or territory where he conducts his specialty practice.

c. He must have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years prior to application for examination by this Board.

d. He must have satisfactorily completed an internship of at least 1 year in a hospital approved for intern training by the American Osteopathic Association or satisfactory to this Board. If graduated in 1942 or prior thereto, the applicant must have had training of value equivalent to that of an acceptable internship as determined by this Board.

e. He must be able to show evidence of conformity to the standards set in the Code of Ethics of the American Osteopathic Association.

f. A period of not less than 3 years of special training related to the specialty shall be required, after the required 1 year of internship, or its equivalent, but not necessarily immediately following the internship nor running 3 consecutive years, except that in the case of applicants who graduated prior to 1946, this Board may modify the requirement of 3 years of special training by allowing a credit of 1 year of special training for each 5 years of specialty practice for a total credit of not to exceed 2 years of the requirement, but in no case may such an applicant graduated prior to 1946 be accepted for examination without at least 1 year of special training as defined. This training shall include:

(i) Advanced study in the sciences: Anatomy, physiology, biochemistry, pathology, pharmacology, and such other basic sciences as are necessary to a mature and comprehensive understanding of the prevention, diagnosis, therapy, and management of the disorders involved in the specialty.

A minimum of 100 clock hours shall be required in the basic science training programs, subdivided as follows:

(a) A minimum of 10 clock hours each in the following subjects: Anatomy, physiology, biochemistry, pathology, and pharmacology.

(b) The remainder of the minimum required hours may be taken in any of the subjects mentioned or in other basic sciences related to the specialty.

(ii) Active experience in diagnosis and treatment in such amount and diversity that it will assure adequate training in the specialty.

The special training may be provided in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board, or by serving an acceptable assistantship to or studying under the preceptorship of a qualified specialist in this field, supplemented by prescribed study conforming to a plan approved by this Board.

The minimum 3-year period of special training may be made up of varying combinations of the several types mentioned above, as determined by this Board.

It shall be this Board's duty to assist applicants in determining a suitable program of special training, but it is not this Board's function to supply the training.

This Board will recognize as proper institutions for securing this training those institutions which are approved for special training by the approving agencies of the American Osteopathic Association, together with such others as this Board may determine to possess facilities and personnel adequate to provide proper training.

g. He must practice as a specialist in his field for a period of at least 2 years subsequent to the minimum 3 years of special training as defined by this Board prior to being examined.

h. Percentage of practice in the field covered by this specialty shall be not less than 60 per cent. The applicant must supply a notarized affidavit of the foregoing.

i. The applicant shall furnish an affidavit of having been first assistant to a qualified specialist in the management of not less than 50 pathological pelvic deliveries as encountered during his assistantship, and 200 cases of a gynecological nature.

j. The applicant must show notarized documented evidence of handling, on his own responsibility, not less than 25 pathological pelvic deliveries and such other nonpathological deliveries as were encountered in the practice of the specialty.

k. The applicant must show notarized documented evidence of handling, as a responsible physician, 100 gynecological cases in which he shall have (a) made a complete examination, including laboratory studies; (b) determined a diagnosis; (c) recommended a major surgical or nonsurgical treatment; (d) observed or participated in a surgical correction of a pathology found, or performed the nonsurgical treatment and (e) followed the patient through her convalescence to recovery. A reasonable proportion of surgical and nonsurgical cases shall be included.

l. The applicant shall send to the Secretary-Treasurer of this Board authentic copies of 15 pathological obstetrical case records, and 10 complicated nonsurgical gynecological case records. The applicant must file an affidavit certifying that the records are exact copies of authentic records in his files, and that he personally cared for the cases presented. All records must be presented in printed form. The obstetrical case records must include a history, physical examination, routine and special laboratory examinations, antepartum, labor, delivery, and postpartum progress records of sufficient quality to clearly reflect the detailed management of the case to the time of dismissal. The gynecological case records shall include history, physical examination, routine and special laboratory examinations, diagnosis, detailed treatment notes, progress notes, and case summary.

m. Thesis:

1. The applicant shall submit a thesis which shall be a report of an original investigation in the specialty field by the applicant or a review of the literature to which the applicant shall add personal experiences referable to the subject matter. This Board may accept a previously published article on a subject in the specialty fields covered by this Board. Grading of the article will be based on English composition and neatness as well as upon the subject matter.

2. Unacceptable Case Records and Thesis

This Board reserves the right to refuse for credit any case record or records or any thesis if, in its judgment, they do not conform to the standards previously mentioned.

n. Following satisfactory compliance with the prescribed requirements as to special training and specialty practice, the applicant shall be required to pass appropriate examinations planned to evaluate his understanding of the scientific bases of the problems involved in the specialty, his familiarity with the current advances in the specialty, the possession of sound judgment and of a high degree of skill in the diagnostic and therapeutic procedures involved in the practice of his specialty. Oral, written, and clinical examinations shall be conducted and required in the case of each applicant. The members of this Board shall personally review, if not perform, the grading of each written examination. The conduct of the clinical examinations may be delegated to committees of not fewer than two individuals maturely qualified in the specialty.

o. Modification of requirements:

In the case of any applicant who was engaged in the practice of his specialty by 1939, or prior thereto, and who graduated prior to 1936, and who meets the general requirements stated in Article IV, Section 1, (a), (b), (c), (d), and (e) of the Bylaws of this Board, and has maintained appropriate activity in American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgradute work, contributions to professional literature, or otherwise has indicated his consistent interest and competence in his specialty field, the requirements of Article IV, Section 1, paragraphs (f) and (h) of the Bylaws of this Board may be waived and he may be recommended for certification after passing an appropriate oral and/or clinical examination.

Obstetrical-Gynecological Surgery:

The applicant must first receive his certification in obstetrics and gynecology by examination from the American Osteopathic Board of Obstetrics and Gynecology. The American Osteopathic Board of Obstetrics and Gynecology shall recommend to the American Osteopathic Board of Surgery the applicant (who has already been certified in obstetrics and gynecology) for examination in obstetrical-gynecological surgery.

The applicant shall file a regular application with the American Osteopathic Board of Surgery stating his request to be certified in obstetrical-gynecological surgery.

If successful in the examination before the American Osteopathic Board of Surgery, the American Osteopathic Board of Surgery shall recommend the applicant for certification in obstetrical-gynecological surgery to the Advisory Board for Osteopathic Specialists according to the regular procedure.

A special certificate shall be issued in obstetrical-gynecological surgery by the American Osteopathic Board of Surgery, signed by the Chairman and Secretary-Treasurer of the American Osteopathic Board of Surgery, Chairman and Secretary-Treasurer of the American Osteopathic Board of Obstetrics and Gynecology, and the Executive Secretary of the American Osteopathic Association.

For requirements, see American Osteopathic Board of Surgery.

AMERICAN OSTEOPATHIC BOARD OF OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY

Preston J. Stack, Chairman, Los Angeles
J. Ernest Leuzinger, Vice Chairman, Philadelphia
Lyman A. Lydic, Dayton, Ohio
C. L. Attebery, Kirksville, Mo.
C. M. Mayberry, East Liverpool, Ohio
Ralph S. Licklider, Secretary-Treasurer, Columbus, Ohio

DEFINITION OF SPECIALTY PRACTICE

Sec. 1. For the purpose of the operation of this Board, the following divisions of practice are defined:

(a) The practice of ophthalmology shall consist of the diagnosis and treatment of the disorders and diseases of the eye and its adnexa, including refraction.

(b) The practice of otorhinolaryngology shall include the diagnosis and treatment of disorders and diseases of the ear, nose, and throat. It may include such other specialties as have a definite relationship to otorhinolaryngology.

(c) The practice of orofacial plastic surgery shall include the diagnosis, medical and surgical treatment and repair of developmental, congenital and acquired deformities of the mouth and face, to include the entire area of the head and neck.

REQUIREMENTS FOR CERTIFICATION

Sec. 1. To be eligible to receive a certificate of specialization from the American Osteopathic Board of Ophthalmology and Otorhinolaryngology the applicant must meet all of the following minimum requirements, excepting as provided in Section 3 and Section 4 of this Article:

(a) He must be a graduate of an approved osteopathic college.

(b) He must be licensed to practice in the state or territory where he conducts his specialty practice.

(c) He must have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years immediately prior to application for examination by this Board, and, if successful in passing his examination, he shall remain a member in good standing in the American Osteopathic Association and in his state or divisional society.

(d) He must have satisfactorily completed an internship of at least 1 year in a hospital approved for intern training by the American Osteopathic Association, or satisfactory to the American Osteopathic Board of Ophthalmology and Otorhinolaryngology. If the applicant was graduated in 1942, or prior thereto, he must furnish evidence of training of equal value satisfactory to the American Osteopathic Board of Ophthalmology and Otorhinolaryngology.

(e) He shall be, and if certified continue to be, favorably known and recognized in his community and by his clientele as an osteopathic physician who is specializing in the practice of ophthalmology and/or otorhinolaryngology, in orofacial plastic surgery, or a specialty coming under the jurisdiction of this Board. He must be able to show evidence of conformity to the standards set in the Code of Ethics of the American Osteopathic Association.

(f) He must provide notarized documentary evidence of a minimum of 3 years of special training in ophthalmology and/or otorhinolaryngology, or in orofacial plastic surgery, after the required 1 year of internship, or its equivalent, but not necessarily running 3 consecutive years. In the case of applicants who graduated prior to 1946, this Board may modify the requirement of 3 years of special training by allowing a credit of 1 year of special training for each 5 years of specialty practice, for a total of not to exceed 2 years of the requirement, but in no case may such applicant be accepted for examination without at least 1 year of special training. This training shall include:

(i) Advanced study in the sciences; Anatomy, physiology, biochemistry, pathology, pharmacology, and such other basic sciences as are necessary to a mature and comprehensive understanding of the prevention, diagnosis, therapy, and management of the disorders involved in the specialty.

(ii) Active experience in the diagnosis and treatment in such amount and diversity that it will assure adequate training in the specialty.

The special training may be provided in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board, or by serving an acceptable assistantship to or studying under the preceptorship of a qualified specialist in this field, supplemented by prescribed study conforming to a plan approved by this Board.

The minimum 3-year period of special training may be made up of varying combinations of the several types mentioned above, as determined by this Board.

(a) The training may consist of a 5-year assistantship to a certified specialist approved by this Board and by the approving agencies of the American Osteopathic Association, during which time he shall have assisted in at least 300 major surgical operations and shall have devoted at least 50 per cent of his time to the training program, followed by at least 2 years of successful specialty practice.

(b) The training may consist of 3 years of full-time assistantship to a certified specialist approved by this Board, and by the approving agencies of the American Osteopathic Association, in both hospital and office practice, followed by at least 2 years of successful specialty practice.

(c) The training may consist of an approved hospital residency in the specialty of not less than 2 years' time, followed by 2 years' assistantship to a certified specialist approved by this Board and 2 years of successful specialty practice.

It shall be this Board's duty to assist applicants in determining a suitable program of special training, but it is not this Board's function to supply the training. This Board will recognize as proper institutions for securing this training those institutions which are approved for special training by the approving agencies of the American Osteopathic Association, together with such others as this Board may determine to possess facilities and personnel adequate to provide proper training.

(g) He shall provide notarized documentary evidence that he has performed 100 major surgical operations in which he was the responsible surgeon.

(h) He shall present detailed case records acceptable to the American Osteopathic Board of Ophthalmology and Otorhinolaryngology of twenty-five major procedures in which he was the responsible surgeon, and handled within the past 5 years. These records shall include history, physical examination, routine laboratory work, special laboratory work, preoperative diagnosis, detailed findings, detailed operative procedure, postoperative diagnosis, the progress notes, gross and histological description and diagnosis of excised tissue, and a case summary.

(i) An applicant for certification by this Board who graduated prior to 1936, and who was engaged in the practice of his specialty by 1939 or prior thereto, and who meets the general requirements stated in Section 1, (a), (b), (c), and (d), and who has maintained appropriate activity in the American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgraduate work, contributions to professional literature, or otherwise has indicated his consistent interest and competence in his specialty, the requirements of this article Section 1, paragraphs (f) and (h) may be waived, and he may be recommended for certification after passing an appropriate oral and/or clinical examination.

(j) As a primary prerequisite for certification in a specialty under the jurisdiction of this Board, the applicant must show evidence of a well-developed understanding of the osteopathic principles and a demonstrated ability to apply these principles.

AMERICAN OSTEOPATHIC BOARD OF PATHOLOGY

Robert P. Morhardt, Chairman, South Pasadena, Calif.
Dorsey A. Hoskins, Vice Chairman, Kansas City, Mo.
Otterbein Dressler, Detroit
William J. Loos, Detroit
Grover C. Stukey, Port Arthur, Tex.
Norman W. Arends, Secretary-Treasurer, Huntington Woods, Mich.

DEFINITION OF SPECIALTY PRACTICE

Sec. 1. For the purpose of the operation of this Board the following divisions of practice are defined:

(a) The practice of clinical pathology shall consist of and include all phases of general clinical laboratory work, hematology, bacteriology, mycology, parasitology, serology, toxicology, and applied biochemistry.

(b) The practice of pathology shall consist of and include surgical pathology, autopsy surgery, histopathology and clinical pathology.

REQUIREMENTS FOR CERTIFICATION

Each applicant for examination for certification in clinical pathology or pathology, or any specialty coming under the jurisdiction of the American Osteopathic Board of Pathology, must present evidence that he has met the following minimum requirements.

a. Educational qualifications

1) An applicant must be a graduate of an osteopathic college approved by the American Osteopathic Association.

2) Internship

a) One year internship, preferably a rotating internship, in a hospital approved for intern training by the American

Osteopathic Association, or under exceptional circumstances other equivalent institutional training for a period of 1 year satisfactory to this Board.

b) An internship of 9 months shall constitute a recognized full internship if served during World War II as prescribed by the Selective Service Board and when such internships were recognized by the American Osteopathic Association as full internships.

c) If graduated in 1942 or prior thereto, the applicant must have had experience and/or training equivalent to that of an acceptable internship, as determined by this Board.

3) Special training

a) Pathology

A period of not less than 3 years of special training is required, after the required 1 year of internship or its equivalent as defined above (a, 2)), but not necessarily immediately following the internship nor running 3 consecutive years.

b) Clinical pathology

A period of not less than 3 years of special training is required, after the required 1 year of internship or its equivalent as defined above (a, 2), but not necessarily immediately following the internship nor running 3 consecutive years.

c) Institutions approved for special training

(1) This Board shall accept special training in pathology or clinical pathology in institutions which meet the Board's requirements and are approved for the specific special training by the approving agencies of the American Osteopathic Association.

(2) This Board requires that the laboratory be under the personal direction of a specialist in pathology (or for clinical pathology, a specialist in clinical pathology) recognized by this Board as capable of proper and thorough teaching of the trainee. He need not be certified but certification in the specialty is preferable.

(3) The laboratory, to be satisfactory, must have an annual minimal volume as follows:

2,000 determinations in hematology;
1,000 determinations in blood chemistry;
1,500 determinations in bacteriology, mycology, parasitology and serology;
2,000 determinations in urinalyses, basal metabolism tests, electrocardiographs, gastric analyses, etc., and general miscellaneous laboratory work.

In addition, for pathology, the following minimal volume is required:

1,000 surgical-case tissues;
Postmortem examination of 35 per cent of the deaths in the hospital. A postmortem examination constitutes at least a thorough examination of the trunk in selected cases, additional explorations of the head, extremities, spinal cord, etc.

(4) The laboratory must have an adequate library in the specialty and in related sciences and fields of practice available to the trainee.

(5) The approval of the laboratory is on an annual basis.

(6) If a laboratory loses its status of approval, a trainee in that laboratory shall be given credit for the time spent in special training only during its period of approval.

d) Programs of special training

(1) The special training program must meet the minimum requirements established by the American Osteopathic Board of Pathology.

(2) The special training program must be approved by the Bureau of Hospitals if the trainee is classified as serving a residency.

(3) The special training program must be approved by the Committee on Accreditation of Postgraduate Training of the Bureau of Professional Education and Colleges if it is other than a residency. This program may be:

i) Fellowships: a graduate training program of a recognized college of osteopathy,

ii) Preceptorships: training programs of a qualified individual,

iii) Miscellaneous: other recognized postgraduate courses given by clinics, colleges, etc.

(4) Residency training, fellowship training, etc., may not be carried out simultaneously in the same laboratory.

(5) The program must meet the requirements to thoroughly train the individual in clinical pathology and/or pathology in all of the branches as defined in 1, a), b), above.

(6) This Board requires the following minimum requirements for approval of a program:

(a) Pathology

This special training shall include:

1. One year's clinical pathology training subdivided as follows:
   i) 3 months' hematology service;
   ii) 3 months' blood chemistry service;
   iii) 3 months' bacteriology, mycology, parasitology, and serology service;
   iv) 3 months' urinalyses and miscellaneous tests and general laboratory work service.
2. Two years' tissue pathology and autopsy surgery

(b) Clinical pathology

This special training shall include:

1. One years' service embracing the four divisions as stated above in (6), (a), 1;
2. Two years in the laboratory.

(c) A year is defined as 50 weeks of a minimum of 40 hours per week.

(d) Attendance at periodic clinicopathologic seminars and/or submission of original theses, the number and character to be determined by the trainer.

e) Status of the trainee

(1) A resident training program comes under the jurisdiction of the Bureau of Hospitals of the American Osteopathic Association. It is defined as a full-time training period appropriate to the specialty and of not less than 1 years' duration spent in an approved osteopathic hospital.

(2) Fellowship training programs come under the jurisdiction of the Committee on Accreditation of Postgraduate Training of the Bureau of Professional Education and Colleges.

(3) An individual may train as a resident or as a Fellow as long as the requirements of the special training program are fulfilled in an institution approved for the special training. Furthermore, a combination of both types may be served if desired or necessary to secure adequate training and may be served in more than one approved laboratory and under more than one qualified specialist.

(4) The trainee must register with this Board within 30 days from the time he begins the special training in his specialty. Forms for registration are available from the Secretary-Treasurer of this Board.

f) Modifications of requirements

The requirements of Article IV, Section 1, f) and h) of the Bylaws may be waived and the applicant recommended for certification after passing an appropriate oral and/or clinical examination, if the applicant was engaged in the practice of his specialty by 1939, or prior thereto, and who graduated prior to 1936, and who meets the general requirements as follows:

(1) He must be a graduate of an approved osteopathic college.

(2) He must be licensed to practice in the state or territory where he conducts his specialty practice.

(3) He must have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years immediately prior to application for examination.

(4) He must have satisfactorily completed an internship of at least 1 year in a hospital approved for intern training by the American Osteopathic Association or satisfactory to this Board. If graduated in 1942 or prior thereto, the applicant must have had training of value equivalent to that of an acceptable internship as determined by this Board.

(5) He must be able to show evidence of conform-

ity to the standards set in the Code of Ethics of the American Osteopathic Association.

(6) He must have maintained appropriate activity in American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgraduate work, contributions to professional literature or otherwise has indicated his consistent interest and competence in his specialty.

g) Modification of requirements by previous commitments by this Board.

In the case of applicants to whom this Board has made definite commitments that a particular program which commenced prior to December 15, 1946, would meet the requirements for admission to examination for certification, the Board may waive such requirements of Article IV, Section 1, paragraphs d), f), and g) of the Bylaws as would prevent the admission of such applicants to examination. The requirements of the Bylaws in force at the time the commitments were made shall apply.

h) Modification of requirements for those who have engaged in specialty practice of pathology or clinical pathology.

If the applicant graduated prior to 1946, this Board may modify the specific requirement of special training by allowing a credit of 1 year of special training for each 5 years of specialty practice for total credit of not to exceed 2 years of the requirement. In no case may such applicant be accepted for examination without at least 1 year of special training as defined.

b. Years of specialty practice

In addition to completing the required special training, the applicant shall have engaged in specialty practice in his field for not less than 2 years before admission to the examination. Specialty practice is defined as a minimum of 90 per cent of the time occupied in professional practice in the specialty, which may include active specialty practice, teaching of pathology, or administration of a department of pathology or research. An affidavit in regard to the above may be required by the Credentials Committee of this Board in evaluating the applicant's application.

c. Membership requirements

The applicant must have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years immediately prior to application for examination by this Board.

d. Licensure requirement

He must be licensed to practice in the state or territory where he conducts his specialty practice.

e. Code of Ethics requirement

He must be able to show evidence of conformity to the standards set in the Code of Ethics of the American Osteopathic Association.

f. Following satisfactory compliance with the prescribed requirements as to special training and specialty practice, the applicant shall be required to pass appropriate examinations planned to evaluate his understanding of the scientific bases of the problems involved in the specialty, his familiarity with the current advances in the specialty, the possession of sound judgment and of a high degree of skill in the diagnostic and therapeutic procedures involved in the practice of the specialty. Oral, written, and clinical examinations shall be conducted and required in the case of each applicant. This Board may conduct the oral and written examinations at the end of the required 3 years of special training, but shall not conduct the practical or clinical examination until the required 2 years of specialty practice have been completed. The members of this Board shall personally review, if not perform, the grading of each written examination. The conduct of the clinical examinations may be delegated to committees of not fewer than two individuals maturely qualified in the specialty. A full description of the method of conducting the examination shall be formulated in this Board's Regulations and Requirements, and provision for re-examination shall be made.

AMERICAN OSTEOPATHIC BOARD OF PEDIATRICS

William S. Spaeth, Chairman, Philadelphia
Mary E. Golden, Vice Chairman, Des Moines, Iowa
Ruth E. Tinley, Philadelphia
James M. Watson, Los Angeles
H. Mayer Dubin, Secretary-Treasurer, Los Angeles

DEFINITION OF SPECIALTY PRACTICE

The practice of pediatrics shall consist of and include the utilization of all of those procedures necessary to the study and the management of the care of infants and children, as well as the prevention, diagnosis, and treatment of the diseases of infants and children from birth up to the average age of sexual maturity.

REQUIREMENTS FOR CERTIFICATION

Each applicant for certification under the jurisdiction of this Board must present evidence that he has met the following minimum requirements:

1. He shall have been graduated from an approved osteopathic college for at least 6 years prior to consideration of his application by the American Osteopathic Board of Pediatrics.

2. He must be licensed to practice in the state or territory where he conducts his specialty practice.

3. He must have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years immediately prior to making application for examination by this Board.

4. He shall be, and if certified continue to be, favorably known and recognized in his community and by his clientele as an osteopathic physician who is specializing in the practice of pediatrics and shall publicize his osteopathic affiliation in an ethical manner.

5. He must be able to show evidence of conformance to the standards set in the Code of Ethics of the American Osteopathic Association.

6. He shall have satisfactorily completed an internship of at least 1 year in a hospital approved for intern training by the American Osteopathic Association or other equivalent institutional training satisfactory to this Board. If the applicant was graduated in 1942 or prior thereto, he must have had training of value equivalent to that of an acceptable internship as determined by this Board.

7. He shall provide notarized documentary evidence of at least 3 years of special training related to the specialty after the required 1 year of internship or its equivalent, but not necessarily immediately following the internship nor running 3 consecutive years. In the case of applicants graduated prior to 1946, the Board may modify the requirements of 3 years of special training by allowing a credit of 1 year of special training for each 5 years of specialty practice for a total credit not to exceed 2 years of the requirement. In no case may such applicant graduated prior to 1946 be accepted for examination without at least 1 year of special training as defined. This training shall include:

(a) Advanced studies in the sciences: Anatomy, physiology, biochemistry, pathology, pharmacology, bacteriology, immunology, parasitology, and such other basic sciences as are necessary to a mature and comprehensive understanding of the prevention, diagnosis, therapy, and management of the disorders involved in the specialty.

(b) Active experience in such amount and diversity in the practice of this specialty that it will assure adequate training in the specialty. The special training may be provided in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board, or by serving an acceptable assistantship to or studying under the preceptorship of a qualified specialist in this field, supplemented by prescribed study conforming to a plan approved by this Board. The minimum 3-year period of special training may be made up of varying combinations of the several types mentioned above as determined by this Board. It shall be this Board's duty to assist applicants in determining a suitable program of special training, but it is not this Board's function to supply the training. This Board will recognize as proper institutions for securing the special training those institutions which are approved by the approving agencies of the American Osteopathic Association together

with such others as this Board may determine to possess facilities and personnel adequate to provide proper training.

8. Subsequent to completing the required special training, the applicant shall have engaged in specialty practice for not less than 2 years before admission to examination. To be considered as engaging in a specialty practice, the applicant shall devote 60 per cent or more of his time to the practice of pediatrics.

9. Following satisfactory compliance with the prescribed requirements as to special training and specialty practice, the applicant shall be required to pass appropriate examinations planned to evaluate his understanding of the scientific bases of the problems involved in this specialty, his familiarity with the current advances in the specialty field, and the possession of sound judgment and of a high degree of skill in the diagnostic and therapeutic procedures involved in the practice of the specialty. Oral, written, and clinical examinations shall be conducted and required in the case of each applicant.

10. In the case of any applicant who was engaged in the practice of his specialty by 1939 or prior thereto and who graduated prior to 1936, and who meets the general requirements as stated in Article IV, Section 1, (a), (b), (c), (d), and (e), of the Bylaws, and has maintained appropriate activity in American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgraduate work, contributions to professional literature, or otherwise has indicated his consistent interest and competence in his specialty, the requirements of Article IV, Section 1, (f) and (h) of the Bylaws may be waived, and he may be recommended for certification after passing an appropriate oral and/or clinical examination.

11. Each applicant for certification coming within the jurisdiction of this Board shall prepare and submit for publication at least three articles on pediatrics during the 3 years of special training and one article in each of the 2 years of specialty practice and shall forward copies of these articles to the Secretary-Treasurer of the American Osteopathic Board of Pediatrics.

AMERICAN OSTEOPATHIC BOARD OF PHYSICAL MEDICINE AND REHABILITATION

William W. W. Pritchard, Chairman, Pasadena, Calif.
Wallace M. Pearson, Vice Chairman, Kirksville, Mo.
Robert C. Ruenitz, Secretary-Treasurer, Los Angeles, Calif.

DEFINITION OF SPECIALTY PRACTICE

The practice of physical medicine and rehabilitation shall consist of and include the use of all medical measures, especially those in the physical field, which will hasten the recovery and restoration of the ill or handicapped to the fullest physical, mental, social, vocational, and economic usefulness of which he or she is capable.

REQUIREMENTS FOR CERTIFICATION

To be eligible to receive a certificate of specialization from this Board, the applicant must meet all of the following minimum requirements, excepting as provided in Article IV, Section 2 of the Bylaws of this Board:

a. He must be a graduate of an approved osteopathic college.

b. He must be licensed to practice in the state or territory where he conducts his specialty practice.

c. He must have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years prior to application for examination by this Board.

d. He must have satisfactorily completed an internship of at least 1 year in a hospital approved for intern training by the American Osteopathic Association or satisfactory to this Board. If graduated in 1942 or prior thereto, the applicant must have had training of value equivalent to that of an acceptable internship as determined by this Board.

e. He must practice as a specialist in his field for a period of at least 2 years subsequent to the minimum 3 years of special training.

f. Percentage of practice in the field covered by this specialty shall not be less than 60 per cent. An affidavit of proof of practice may be required by this Board from the applicant.

g. Number of case records

1. The applicant must present evidence of handling on his own responsibility not less than 200 cases applicable to this specialty. This evidence must include: history, structural examinations, general physical examination, record of special muscle and nerve testings where applicable, treatment prescribed, and progress notes of the case, including termination of treatment or at the latest date patient was observed.

2. Documentary evidence of the above may be requested by this Board.

3. These records become the property of this Board and shall be placed on file with the Secretary-Treasurer for a minimum of 3 years.

h. Thesis

1. The applicant shall submit reprints or copies of representative articles which have been published or have been prepared for publication. The thesis, a monograph, shall be an original investigation in the specialty field as conducted by the applicant or a review of the literature to which the applicant shall add personal experiences referable to the subject matter.

2. This Board shall submit to the applicant three titles as suggestions for the monograph. If these are not acceptable to the applicant, the applicant may submit a title of his choice to the Board for approval.

3. The title of the thesis must be approved by this Board and the title shall be recorded in the office of the Secretary-Treasurer.

4. The form of the thesis shall follow a stylebook of the applicant's choosing. The author and title of the stylebook shall be included in the bibliography.

5. Sufficient copies shall be submitted to the Secretary-Treasurer of this Board to permit distribution of the thesis to the members of the Examination Committee.

i. Training

A period of not less than 3 years of special training related to the specialty shall be required, after the required 1 year of internship or its equivalent, but need not immediately follow the internship, nor run 3 years consecutively. In the case of applicants who graduated prior to 1946, this Board may modify the requirements of 3 years' special training by allowing a credit of one year of special training for each 5 years of specialty practice for a total credit of not to exceed 2 years of the requirement, but in no case may such an applicant graduated prior to 1946 be accepted for examination without at least 1 year of special training.

This training shall include:

1. Advanced study in the sciences: Anatomy, physics, physiology, biochemistry, pathology, pharmacology, and such other basic sciences as may be required by this Board.

2. Active experience in the diagnosis and treatment in such amount and diversity that it will assure adequate training in the specialty.

3. The clinical aspects of physical medicine:

a. Thermotherapy

Clinical applications of heat and cold, both local and general.

b. Hydrotherapy

Methods of employing hydrotherapeutic devices and procedures.

c. Electrotherapy

Clinical, diagnostic, and therapeutic uses of the constant current, interrupted current, sinusoidal current, and high frequency current.

d. Radiation therapy

Diagnostic and therapeutic uses of ultra-violet and infra-red radiation. (Therapeutic applications of roentgen rays and atomic radiation are not included in the field of physical medicine and rehabilitation.)

e. Kinesitherapy

Clinical uses in diagnosis and treatment by exercise.

f. Occupational therapy

Indications for and clinical applications of the various types.

g. Rehabilitation

The diagnostic and therapeutic use of convalescent reconditioning and rehabilitation of the seriously disabled person.

The special training may be provided in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board, or by serving an acceptable assistantship to or studying under the preceptorship of, a qualified specialist in this field, supplemented by prescribed study conforming to a plan approved by this Board.

The minimum 3-year period of special training may be made up of varying combinations of several types mentioned above, as determined by this Board.

It shall be this Board's duty to assist applicants in determining a suitable program of special training, but it is not this Board's function to supply the training.

This Board will recognize as proper institutions for securing this training those institutions which are approved for special training by the approving agencies of the American Osteopathic Association, and such others as this Board may determine to possess facilities and personnel adequate to provide proper training.

j. Modification of Requirements

In the case of any applicant who was engaged in the practice of his specialty by 1939 or prior thereto, and who graduated prior to 1936, and who meets the general requirements stated in Article IV, Section 1, (a), (b), (c), (d), and (e) of the Bylaws of this Board, and has maintained appropriate activity in the American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgraduate work, contributions to professional literature or otherwise has indicated his consistent interest and competence in his specialty field, the requirements of Article IV, Section 1, paragraphs (f) and (h) of the Bylaws of this Board, may be waived and he may be recommended for certification after passing an appropriate oral and/or clinical examination.

k. Following satisfactory compliance with the prescribed requirements as to special training and specialty practice, the applicant shall be required to pass appropriate examinations planned to evaluate his understanding of the scientific bases of the problems involved in the specialty, his familiarity with the current advances in the specialty, the possession of sound judgment and of a high degree of skill in the diagnostic and therapeutic procedures involved in the practice of the specialty. Oral, written, and clinical examinations shall be conducted and required in the case of each applicant. This Board may conduct the oral and written examinations at the end of the required 3 years of special training, but shall not conduct the practical or clinical examination until the required 2 years of specialty practice have been completed. The members of this Board shall personally review, if not perform, the grading of each written examination. The conduct of the clinical examinations may be delegated to committees of not fewer than two individuals maturely qualified in the specialty. A full description of the method of conducting the examination shall be formulated in this Board's Regulations and Requirements, and provision for re-examination shall be made.

AMERICAN OSTEOPATHIC BOARD OF PROCTOLOGY

Carl S. Stillman, Jr., Chairman, San Diego, Calif.
Frank D. Stanton, Vice Chairman, Boston
John M. Spencer, St. Joseph, Mo.
John W. Orman, Tulsa, Okla.
Carlton M. Noll, Secretary-Treasurer, Denver

DEFINITION OF SPECIALTY PRACTICE

For the purpose of clarification the following division of specialty practice is defined:

a. The practice of proctology shall consist of and include the treatment of diseases and conditions originating within or affecting the anus, rectum, perianal and perirectal areas, and related or complicating conditions not including the use of major surgery.

REQUIREMENTS FOR CERTIFICATION

To be eligible to receive a certificate of specialization from the American Osteopathic Board of Proctology, the applicant must meet all of the following minimum requirements except as provided in Article IV, Section 2, of the Bylaws of the American Osteopathic Board of Proctology.

a. He must have been graduated from an approved osteopathic college at least 5 years prior to application.

b. He must be licensed to practice in the state or territory where he conducts his specialty practice.

c. He must have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years immediately prior to application for examination by this Board.

d. He must have satisfactorily completed an internship of at least 1 year in a hospital approved for intern training by the American Osteopathic Association or other equivalent institutional training satisfactory to this Board. If graduated in 1942 or prior thereto, the applicant must have had training of value equivalent to that of an acceptable internship as determined by this Board.

e. He shall be, and if certified continue to be, favorably known and recognized in his community and by his clientele as an osteopathic physician who is specializing in the practice of proctology and shall publicize his osteopathic affiliation in an ethical manner.

f. He must be able to show evidence of conformity to the standards set in the Code of Ethics of the American Osteopathic Association.

g. A period of not less than 3 years of special training related to the specialty shall be required, after the required 1 year internship, or its equivalent, but not necessarily immediately following the internship nor running 3 consecutive years, except that in the case of applicants who graduated prior to 1946, the Board may allow the requirement of 3 years of special training by allowing a credit of 1 year of special training for each 5 years of specialty practice for a total credit of not to exceed 2 years of the requirement, but in no case may an applicant graduated prior to 1946 be accepted for examination without at least 1 year of special training as defined. This training shall include:

(i) Advanced study in the sciences: Anatomy, physiology, biochemistry, pathology, pharmacology, and such other basic sciences as are necessary to a mature and comprehensive understanding of the prevention, diagnosis, therapy, and management of the disorders involved in the specialty.

The minimum requirement in the basic sciences shall be 100 clock hours subdivided as follows:

Anatomy 10 hours; physiology 10 hours; biochemistry 10 hours; pathology 10 hours; pharmacology 10 hours; bacteriology and parasitology 10 hours.

The balance of 40 hours shall include such other basic sciences as this Board may require from time to time.

(ii) Active experience in diagnosis and treatment in such amount and diversity that it will assure adequate training in the specialty.

h. He must practice as a specialist in his field for a period of at least 2 years subsequent to the minimum 3 years of special training.

i. Training Requirements:

(1) Active experience in the diagnosis and treatment in such amount and diversity that it will assure adequate training in the specialty. The special training may be provided in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board, or by serving an acceptable assistantship to or studying under the preceptorship of a qualified specialist in this field, supplemented by prescribed study conforming to a plan approved by this Board.

The minimum 3-year period of special training may be made up of varying combinations of several types mentioned above, as determined by this Board. It shall be this Board's duty to assist applicants in determining a suitable program of special training, but it is not this Board's function to supply the training. This Board will recognize as proper institutions for securing this training those institutions which are approved for special training by the approving agencies of the American Osteopathic Association, together with such others as this Board may determine to possess facilities and personnel adequate to provide proper training.

j. Following satisfactory compliance with the prescribed requirements as to special training and specialty practice, the applicant shall be required to pass appropriate examinations planned to evaluate his understanding of the scientific bases of the problems involved in the specialty, his familiarity with the current advances in the specialty, the possession of sound judgment, and a high degree of skill in the diagnostic and therapeutic procedures involved in the practice of the specialty. Oral, written, and clinical examinations shall be conducted and required in the case of each applicant. This Board of specialty certification may conduct its oral and written examinations at the end of the required 3 years of special training, but shall not conduct the practical or clinical examination until the required 2 years of specialty practice have been completed. The members of this Board shall personally review, if not perform, the grading of each written examination. The conduct of the clinical examinations may be delegated to committees of not fewer than two individuals maturely qualified in the specialty.

k. In the case of any applicant who has engaged in the practice of his specialty by 1939, or prior thereto, and who graduated prior to 1936, and who meets the general requirements stated in Section 1, (a), (b), (c), (d), and (e) of the Bylaws of this Board, and has maintained appropriate activity in the American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgraduate work, contributions to professional literature, or otherwise has indicated his consistent interest and competence in his specialty, the requirements of Article IV, paragraphs (f) and (h) of the Bylaws of this Board may be waived and he may be recommended for certification after passing an appropriate oral and/or clinical examination.

l. Affidavit:

The applicant shall file with the Secretary-Treasurer of this Board an affidavit in duplicate stating the percentage of his practice that was proctologic in character during the preceding 12-month period, based on the number of patients seen. An applicant must have a minimum of 75 per cent proctologic specialty practice to be eligible for examination.

m. Case Records:

The applicant must file with the Secretary-Treasurer of this Board true and authentic copies of 200 case records of proctologic cases he has cared for within the 4-year interval preceding the filing of his application. The applicant must file an affidavit in duplicate certifying that the records are exact copies of true and authentic records in his files and that he personally cared for the cases represented. The case records and affidavit must reach the Secretary-Treasurer of this Board not later than April 15th for the applicant to be eligible for examination at the following annual meeting of this Board. All records become the property of this Board and are not returnable to the applicant.

n. Scientific Article:

The applicant must file with the Secretary-Treasurer of this Board by April 15th of the year he desires examination, a 1,000-word article on a proctologic subject of his choosing or at the dictates of this Board. This Board may accept a previously published article on a proctologic subject. Grading of this scientific article will be based on English composition and neatness as well as upon the subject matter. Scientific articles must follow the standard in common use for composition in such articles.

AMERICAN OSTEOPATHIC BOARD OF RADIOLOGY

A. H. Witthohn, Chairman, Grand Rapids, Mich.
Clyde C. Henry, Vice Chairman, Denver
Jack H. Grant, Chicago
Burwell S. Keyes, Los Angeles
William L. Tanenbaum, Philadelphia
D. W. Hendrickson, Secretary-Treasurer, Wichita, Kan.

DEFINITION OF SPECIALTY PRACTICE

For the purposes of clarification the following definitions of the divisions of the special fields in radiology are given:

a. The practice of diagnostic roentgenology shall consist of and be confined to the performance and interpretation of diagnostic roentgenological procedures.

b. The practice of roentgenology shall consist of and be limited to the performance and interpretation of diagnostic roentgenological procedures and the use of roentgen rays in therapy.

c. The practice of radiation therapy shall consist of treatment of human disease by the use of roentgen rays, radium, and artificial radioactive substances.

d. The practice of radiology shall consist of and include the performance and interpretation of diagnostic roentgenological procedures and the diagnosis and treatment of human disease by the use of roentgen rays, radium, and artificial radioactive substances.

REQUIREMENTS FOR CERTIFICATION

The American Osteopathic Board of Radiology will accept for certification only osteopathic physicians who are specializing in one of the divisions of radiology coming under the jurisdiction of this Board. The proportion of specialization in practice which is acceptable to the Board may vary according to the size of the community and the conditions under which the applicant practices. The applicant must be engaged in the practice of one of the divisions of radiology coming under the jurisdiction of this Board, i.e., diagnostic roentgenology, roentgenology, radiology, or radiation therapy, and he must present evidence satisfactory to the Board that he is so practicing.

Each applicant for certification under this Board must present evidence that he has met the following minimum requirements:

1. He shall have been graduated from an approved osteopathic college for at least 5 years prior to consideration of his application by this Board.

2. He must have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years immediately prior to making application for examination by this Board.

3. He shall be, and if certified continue to be, favorably known and recognized in his community and by his clientele as an osteopathic physician who is specializing in the practice of one of the divisions of radiology coming under the jurisdiction of this Board and shall publicize his osteopathic affiliations in an ethical manner.

4. He must have satisfactorily completed an internship of at least 1 year in a hospital approved for intern training by the American Osteopathic Association or satisfactory to this Board. If graduated in 1942 or prior thereto, the applicant must have had training of value equivalent to that of an acceptable internship as determined by this Board. The intern year of 9 months as prescribed by the Selective Service Board during World War II shall be acceptable as fulfilling this requirement.

5. He must be licensed to practice in the state or territory where he conducts his specialty practice.

6. He must be able to show evidence of conformity to the standards set in the Code of Ethics of the American Osteopathic Association.

7. He shall provide documentary evidence of at least 3 years of special training related to the specialty for which he is seeking certification, after the required 1 year of internship, or its equivalent, but not necessarily running 3 consecutive years. This Board may modify the requirements of 3 years' special training by allowing a credit of 1 year of special training for each 5 years of specialty practice for a total credit not to exceed 2 years of the requirement, for physicians who graduated prior to 1946, but in no case may such applicant be accepted for examination without at least 1 year of special training as defined. This training shall include:

(a) Advanced study in the sciences: Anatomy, physiology, biochemistry, pathology, pharmacology, and such other basic sciences as are necessary to a mature and comprehensive understanding of the prevention, diagnosis, therapy, and management of the disorders involved in the specialty.

(b) Active experience in such amount and diversity that it will assure adequate training in the specialty.

The special training may be provided in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board, or by serving an acceptable assistantship to or studying

under the preceptorship of a qualified specialist in this field, supplemented by prescribed study conforming to a plan approved by this Board.

The minimum 3-year period of special training may be made up of varying combinations of the several types mentioned above, as determined by this Board. It shall be this Board's duty to assist applicants in determining a suitable program of special training, but it is not this Board's function to supply the training. This Board will recognize as proper institutions for securing this training those institutions which are approved for special training by the approving agencies of the American Osteopathic Association, together with such others as this Board may determine to possess facilities and personnel adequate to provide proper training.

8. In addition to completing the required special training, the applicant shall have engaged in specialty practice in his field for not less than 2 years before admission to examination.

9. Following satisfactory compliance with the prescribed requirements as to special training and specialty practice, the applicant shall be required to pass appropriate examinations planned to evaluate his understanding of the scientific bases of the problems involved in the specialty, his familiarity with the current advances in the specialty, and the possession of sound judgment and of a high degree of skill in the diagnostic and therapeutic procedures involved in the practice of the specialty. Oral, written, and clinical examinations shall be conducted and required in the case of each applicant. This Board may conduct the oral and written examinations at the end of the required 3 years of special training, but shall not conduct the practical or clinical examination until the required 2 years of specialty practice have been completed. The members of this Board shall personally review, if not perform, the grading of each written examination. The conduct of the clinical examinations may be delegated to committees of not fewer than two individuals maturely qualified in the specialty. A full description of the method of conducting the examination shall be formulated in this Board's Regulations and Requirements, and provision for re-examination shall be made.

10. In the case of any applicant who was engaged in the practice of his specialty by 1939, or prior thereto, and who graduated prior to 1936, and who meets the general requirements as stated above, and has maintained appropriate activity in American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgraduate work, contributions to professional literature, or otherwise has indicated his consistent interest and competence in his specialty, the requirements of Article IV, Section 1, paragraphs (f) and (h) of the Bylaws may be waived and he may be recommended for certification after passing appropriate oral and/or written clinical examination.

11. Applicants for certification in any of the divisions of radiology shall prepare a thesis upon some phase of radiology as a part of the written examination for certification.

AMERICAN OSTEOPATHIC BOARD OF SURGERY

James M. Eaton, Chairman, Philadelphia
Lucius B. Faires, Vice Chairman, Hollywood, Calif.
Charles L. Ballinger, Toledo, Ohio
Howard A. Graney, Des Moines, Iowa
Don E. Ranney, Grosse Pointe Woods, Mich.
William G. Stahl, Pomona, Calif.
K. George Tomajan, Boston
Ernest G. Bashor, Los Angeles
Arthur M. Flack, Jr., Secretary-Treasurer, Philadelphia

DEFINITION OF SPECIALTY PRACTICE

For the purpose of clarification the following divisions of specialty practice coming under the jurisdiction of the American Osteopathic Board of Surgery are defined.

1. The practice of surgery shall consist of and include that branch of medical science, art, and practice which is concerned with the correction of deformities and defects, the repair of injuries, the diagnosis and care of disease, the relief of suffering, and the prolongation of life by manual and instrumental means.

2. The practice of neurosurgery shall consist of and include that branch of medical science, art, and practice which is concerned with the correction of deformities and defects, the repair of injuries, and the diagnosis and care of diseases of the nervous system by manual and instrumental means.

3. The practice of obstetrical-gynecological surgery shall consist of and include that branch of medical science, art, and practice which is concerned with major operative procedures within the abdomen and pelvis upon the female generative system or structures related thereto, and major plastic procedures upon the vaginal tract by manual and instrumental means.

4. The practice of orthopedic surgery shall consist of and include that branch of medical science, art, and practice which is concerned with the correction of deformities and defects of the bony skeleton and associated structures, the repair of injuries to the bones and joints, and the diagnosis and care of diseases of the bones and joints by manual and instrumental means.

5. The practice of peripheral vascular surgery shall consist of and include that branch of medical science, art, and practice which is concerned with the correction of deformities and defects of the peripheral vessels, the repair of injuries to the peripheral vessels, and the diagnosis and care of peripheral vascular diseases by manual and instrumental means.

6. The practice of plastic surgery shall consist of and include that branch of medical science, art, and practice which is concerned with the repair of injuries and the correction of defects and deformities to the integument, muscles, the bony skeleton and associated structures of the body by manual and instrumental means.

7. The practice of urological surgery shall consist of and include that branch of medical science, art, and practice which is concerned with the correction of deformities and defects, the repair of injuries, and the diagnosis and care of diseases of the urogenital organs by manual and instrumental means.

8. The practice of anesthesiology shall consist of and include that branch of medical science, art, and practice which deals with the use of anesthetic agents, the evaluation of the patient's condition with reference to the use of anesthetic agents, and the care of complications incident to their use.

BASIC REQUIREMENTS FOR CERTIFICATION

The American Osteopathic Board of Surgery shall accept for examination for certification only osteopathic physicians who are specializing in one of the specialty fields coming under the jurisdiction of this Board. The proportion of specialization in practice which is acceptable to this Board may vary according to the size of the community and the conditions under which the applicant practices.

The applicant must be engaged in the practice of surgery or one of the specialty fields coming under the jurisdiction of this Board, i.e., neurosurgery, obstetrical-gynecological surgery, orthopedic surgery, peripheral vascular surgery, plastic surgery, urological surgery, or anesthesiology and he must present evidence to this Board that he is so practicing.

It shall be the policy of the American Osteopathic Board of Surgery to require each applicant to submit a list of his surgical operative procedures (or anesthetic procedures) for the 12-month period immediately prior to making application, naming the type of operation, the hospital in which the operation was performed, the patient's initials, the hospital case number, the pathologist's report, and the case termination.

This list shall be certified by the administrator of the hospital in which the surgeries were performed and shall be accompanied by a supplementary list in which the operative procedures for the year are broken down into segregated totals indicating the number of each type of operative procedure performed by the applicant during the 12-month period. The totals of both lists must agree.

It shall be the policy of this Board to require the Credentials Committee to carefully scrutinize the scope of surgical or anesthetic practice represented in each applicant's report. Unless an applicant presents evidence that the scope of his specialty practice is sufficiently varied and of a major character (as shown by his hospital records) the Credentials Committee shall not recommend him for examination without an examination by one or more certified specialists not located in

the applicant's immediate vicinity who have been appointed by this Board for the purpose of determining the applicant's qualifications.

It shall be the policy of this Board to admit to examination only those surgeons or anesthetists who qualify as recognized specialists in their respective fields in the community in which they conduct their specialty practice.

Each applicant for examination for certification in surgery or one of the surgical specialty fields coming under the jurisdiction of the American Osteopathic Board of Surgery must present evidence that he has met the following minimum requirements:

1. He shall have graduated from an approved osteopathic college at least 7 years prior to consideration of his application by this Board.

2. He shall be licensed to practice in the state or territory where he conducts his specialty practice.

3. He shall have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years immediately prior to making application for examination by this Board.

4. He shall be, and if certified continue to be, favorably known and recognized by his clientele as an osteopathic physician who is specializing in the practice of surgery or a surgical specialty coming under the jurisdiction of this Board and shall publicize his osteopathic affiliations in an ethical manner.

5. He shall be able to show evidence of conformity to the standards set in the Code of Ethics of the American Osteopathic Association.

6. He shall have had an internship of at least 1 year in a hospital acceptable to this Board. The intern year of 9 months as prescribed by the Selective Service Board during World War II shall be acceptable. For physicians who graduated in 1942 or prior thereto this Board may accept other qualifications in lieu of internship where the applicant's surgical experience and ability are beyond question.

7. He shall provide notarized documentary evidence of a minimum of 3 years of formal training subsequent to his internship. This formal training program must be acceptable to this Board and made up, either in whole or in part, of (a) an approved surgical residency; (b) approved preceptorship training programs which commenced after January 1, 1951, shall consist of 3 years for full-time training programs or 5 years for part-time training programs; (c) approved postgraduate work in surgery or the surgical sciences in a program acceptable to this Board. Not more than 1 year's credit shall be given for postgraduate work in approved graduate school programs.

During the period of his formal training, he shall have assisted in the performance of not less than 400 major surgical operations. By "first assistant" is meant one who, in addition to acting as first assistant at the operating table, has participated actively in the preoperative diagnostic procedures and postoperative care.

In qualifying for examination for certification only one credit for first assistantship is allowable for each case done by the surgeon regardless of the number of assistants working at the table.

8. He shall provide evidence to this Board that he has received satisfactory postgraduate training in the surgical sciences, including anatomy, physiology, biochemistry, pathology, and pharmacology.

The applicant who began a specialty training program after January 1, 1951, and who has pursued a training program other than a residency approved by the Board of Trustees of the American Osteopathic Association, shall provide evidence to this Board that he has received satisfactory postgraduate training in the surgical sciences, including anatomy, physiology, biochemistry, pathology, pharmacology, and such other basic sciences as are necessary to a mature and comprehensive understanding of the prevention, diagnosis, therapy, and management of the disorders involved in the specialty.

A minimum of 100 clock hours shall be required in the basic science training programs, subdivided as follows:

a. A minimum of 10 clock hours each in the following subjects: anatomy, physiology, biochemistry, pathology and pharmacology.

b. The remainder of the minimum required hours may be taken in any of the subjects mentioned or in other basic sciences related to the specialty.

Those applicants who qualify must submit acceptable documentary evidence, which may be further investigated at the discretion of this Board, of having pursued this advanced training in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board.

9. He must practice as a specialist in his field for a period of at least 2 years subsequent to the completion of the required minimum of 3 years of special training prior to being examined. He shall provide notarized documentary evidence that he has performed a minimum of 200 major surgical operations upon his own responsibility subsequent to the completion of the minimum required period of formal training.

An applicant's scope of surgical practice must be such that his records will show that he has handled and is capable of handling (as shown by his morbidity, mortality, and end results) major surgical procedures in his respective specialty field. In surgery this would include such procedures as thyroidectomies, gastrectomies, excision of the colon, etc. An applicant whose hospital records show that he has never performed what are commonly referred to as heavy surgical procedures on his own responsibility would not be eligible for admission to examination.

10. He shall demonstrate his fitness to take the examination in any other manner required by this Board.

11. He shall present detailed case records, acceptable to this Board, of 25 major surgical procedures in which he was the responsible surgeon and handled by him as a surgical problem within the past 3 years. These records shall include history, physical examination, routine and special laboratory examinations, preoperative diagnosis, detailed findings, detailed operative procedure, postoperative diagnosis, progress notes, gross and histological description and diagnosis of the excised tissue, and case summary.

12. In the case of any applicant who was engaged in the practice of surgery (or one of the specialty fields of surgery coming under the jurisdiction of this Board) by 1939, or prior thereto, and who graduated prior to 1936, and who meets the general requirements stated in Article IV, Section 1, (a), (b), (c), (d), and (e) of the Bylaws, and who has maintained appropriate activity in American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgraduate work, contributions to professional literature, or otherwise has indicated his consistent interest and competence in his specialty field, the requirements of Article IV, Section 1, (f) and (h) of the Bylaws may be waived and the applicant may be recommended for certification after passing an appropriate oral and/or clinical examination.

NEUROSURGERY

Each applicant for examination for certification in neurosurgery must present evidence that he has met the minimum basic requirements.

1. Under requirement No. 7, he shall have assisted in the performance of not less than 400 major surgical operations, 200 of which must be of a major neurosurgical nature.

2. Under requirement No. 9, he shall have performed a minimum of 200 major surgical operations in which he was the responsible surgeon, 100 of which must be of a major neurosurgical nature.

3. Under requirement No. 11, the case records presented shall consist of 25 major neurosurgical procedures.

4. He shall demonstrate his fitness to take the examination in any other manner required by this Board.

5. If this Board approves his application as to qualifications for examination in neurosurgery, the Secretary-Treasurer shall request the American Osteopathic Board of Neurology and Psychiatry to consider the application and, if acceptable, to examine the applicant in neurology.

6. If the applicant is successful before the American Osteopathic Board of Neurology and Psychiatry, the American Osteopathic Board of Surgery shall be so informed and shall, in turn, examine the applicant in neurosurgery.

7. If the applicant is successful in the examination before the American Osteopathic Board of Surgery, this Board shall

make recommendation for certification to the Advisory Board for Osteopathic Specialists according to the regular procedure.

8. Upon final approval by the Board of Trustees of the American Osteopathic Association, a special certificate shall be issued in neurosurgery by this Board, signed by the Chairman and Secretary-Treasurer of the American Osteopathic Board of Neurology and Psychiatry, in addition to the officers of the American Osteopathic Board of Surgery and the Executive Secretary of the American Osteopathic Association.

OBSTETRICAL-GYNECOLOGICAL SURGERY

Each applicant for examination for certification in obstretical-gynecological surgery must first have received his certification in obstetrics and gynecology by examination by the American Osteopathic Board of Obstetrics and Gynecology. Having been recommended by the American Osteopathic Board of Obstetrics and Gynecology for examination in obstetrical-gynecological surgery, each applicant shall present evidence that he has met the minimum basic requirements.

1. Under requirement No. 7, he shall have assisted in the performance of not less than 400 major surgical operations, 200 of which must be of a major obstetrical-gynecological nature. Not more than 25 per cent of any one surgical procedure shall be permitted for accreditation towards the required number of operative assistantships.

2. Under requirement No. 9, he shall have performed a minimum of 200 major surgical operations in which he was the responsible surgeon, 100 of which must be of a major obstetrical-gynecological nature. Not more than 25 per cent of any one surgical procedure shall be permitted for accreditation towards the required number of operative procedures performed on his own responsibility.

3. Under requirement No. 11, the case records presented shall consist of 25 major obstetrical-gynecological procedures.

4. He shall demonstrate his fitness to take the examination in any other manner required by this Board.

5. If successful in the examination before the American Osteopathic Board of Surgery, this Board shall make recommendation for certification to the Advisory Board for Osteopathic Specialists according to the regular procedure.

6. Upon final approval of the Board of Trustees of the American Osteopathic Association, a special certificate shall be issued in obstetrical-gynecological surgery by this Board, signed by the Chairman and Secretary-Treasurer of the American Osteopathic Board of Obstetrics and Gynecology, in addition to the officers of the American Osteopathic Board of Surgery and the Executive Secretary of the American Osteopathic Association.

Physicians certified in surgery by the American Osteopathic Board of Surgery may apply for change of certification in obstetrical-gynecological surgery, by notifying the Secretary-Treasurer of this Board in writing. Upon receipt of such notification, the Secretary-Treasurer of this Board shall notify the Chairman and Secretary-Treasurer of the American Osteopathic Board of Obstetrics and Gynecology of the request for change in certification.

If no written dissent is received by the Secretary-Treasurer of this Board from the American Osteopathic Board of Obstetrics and Gynecology prior to the next annual meeting of the American Osteopathic Board of Surgery, the Secretary-Treasurer shall submit the request to this Board at its annual meeting. After due investigation, this Board shall act upon the requested change in certification and transmit its recommendation to the Advisory Board for Osteopathic Specialists and the Board of Trustees of the American Osteopathic Association according to the regular procedure.

ORTHOPEDIC SURGERY

Each applicant for examination for certification in orthopedic surgery must present evidence that he has met the minimum basic requirements.

1. Under requirement No. 7, he shall have assisted in the performance of not less than 400 major surgical operations, 200 of which must be of a major orthopedic nature.

2. Under requirement No. 9, he shall have performed a minimum of 200 major surgical operations in which he was the responsible surgeon, 100 of which must be of a major orthopedic nature.

3. Under requirement No. 11, the case records presented shall consist of 25 major orthopedic procedures.

4. He shall demonstrate his fitness to take the examination in any other manner required by this Board.

PERIPHERAL VASCULAR SURGERY

Each applicant for examination for certification in peripheral vascular surgery must present evidence that he has met the minimum basic requirements.

1. Under requirement No. 7, he shall have assisted in the performance of not less than 400 major surgical operations, 200 of which must be of a major peripheral vascular nature.

2. Under requirement No. 9, he shall have performed a minimum of 200 major surgical operations in which he was the responsible surgeon, 100 of which must be of a major peripheral vascular nature.

3. Under requirement No. 11, the case records presented shall consist of 25 major peripheral vascular surgical procedures.

4. He shall demonstrate his fitness to take the examination in any other manner required by this Board.

PLASTIC SURGERY

Each applicant for examination for certification in plastic surgery must present evidence that he has met the minimum basic requirements.

1. Under requirement No. 7, he shall have completed a minimum of 2 years of approved formal surgical training either in an approved residency or an approved full-time preceptorship training program followed by 1 or more years of acceptable training in the specialty field of plastic surgery. He shall have assisted in the performance of not less than 400 major surgical operations, 200 of which must be of a major plastic surgical nature.

2. Under requirement No. 9, he shall have performed a minimum of 200 major surgical operations in which he was the responsible surgeon, 100 of which must be of a major plastic surgical nature.

3. Under requirement No. 11, the case records presented shall consist of 25 major plastic surgical procedures.

4. He shall demonstrate his fitness to take the examination in any other manner required by this Board.

5. A physician certified in surgery who desires to have his certification changed from surgery to plastic surgery shall be required to follow the prescribed procedure.

UROLOGICAL SURGERY

Each applicant for examination for certification in urological surgery must present evidence that he has met the minimum basic requirements.

1. Under requirement No. 7, he shall have assisted in the performance of not less than 400 major surgical operations, 200 of which must be of a major urological nature.

2. Under requirement No. 9, he shall have performed a minimum of 200 major surgical operations in which he was the responsible surgeon, 100 of which must be of a major urological nature.

3. Under requirement No. 11, the case records shall consist of 25 major urological procedures.

4. He shall demonstrate his fitness to take the examination in any other manner required by this Board.

ANESTHESIOLOGY

Each applicant for examination for certification in anesthesiology must present evidence that he has met the following minimum requirements:

1. He shall have graduated from an approved osteopathic college at least 6 years prior to consideration of his application by this Board.

2. He shall be licensed to practice in the state or territory where he conducts his specialty practice.

3. He shall have been a member in good standing of the American Osteopathic Association and of his state or divisional society for a continuous period of at least 3 years immediately prior to making application for examination by this Board.

4. He shall be, and if certified continue to be, favorably known and recognized by his clientele as an osteopathic physi-

cian who is specializing in the practice of anesthesiology and shall publicize his osteopathic affiliations in an ethical manner.

5. He shall be able to show evidence of conformity to the standards set in the Code of Ethics of the American Osteopathic Association.

6. He shall have had an internship of at least 1 year in a hospital acceptable to this Board. The intern year of 9 months as prescribed by the Selective Service Board during World War II shall be acceptable. For physicians who graduated in 1942 or prior thereto, this Board may accept other qualifications in lieu of internship where the applicant's anesthetic experience and ability are beyond question.

7. He shall provide notarized documentary evidence of a minimum of 3 years of formal training subsequent to his internship (or its equivalent). This formal training program must be acceptable to this Board and shall include:

a) A period of at least 1 year of formal training in anesthesiology in a hospital acceptable to this Board and made up of either:

i) an approved residency;

or

ii) an approved full-time preceptorship training program.

b) A period of at least 2 years' special training in anesthesiology subsequent to the required 1 year of internship and the required 1 year of formal training. This special training may be made up, either in whole or in part, of:

i) an approved residency;

ii) approved preceptorship training. Approved preceptorship training programs may be either full-time or part-time. Two years of approved part-time preceptorship training shall be the equivalent of 1 year of full-time preceptorship training.

After the completion of the required 1 year of internship (or its equivalent), the required 1 year of formal training, and 1 year of the required 2 years of special training in anesthesiology, this Board may, at its discretion, accept 2 years of recognized specialty practice in anesthesiology as the equivalent of the third year of special training. In each year of specialty practice, the applicant must have administered at least 250 anesthetics of a diversified nature.

During the minimum 3-year period of formal training, there shall be evidence of sufficient clinical work in anesthesiology with a minimum of 250 anesthetics of a diversified nature administered annually.

For applicants who graduated in 1947 or prior thereto, in lieu of requirement No. 7, Item b), this Board may, at its discretion, accept 2 years of recognized specialty practice in anesthesiology, subsequent to the required 1 year of internship (or its equivalent), and the required 1 year of formal training, as the equivalent of 1 year of special training or 4 years of recognized specialty practice in anesthesiology as the equivalent of 2 years of special training. In each year of specialty practice, the applicant must have administered at least 250 anesthetics of a diversified nature.

8. He shall provide evidence to this Board that he has received satisfactory postgraduate training in the basic sciences, including anatomy, physiology, biochemistry, pathology, toxicology, and pharmacology.

The applicant who began a specialty training program in anesthesiology after January 1, 1951, and who has pursued a training program in anesthesiology other than a residency approved by the Board of Trustees of the American Osteopathic Association shall provide evidence to this Board that he has received satisfactory postgraduate training in the basic sciences, including anatomy, physiology, biochemistry, pathology, toxicology, and pharmacology.

A minimum of 100 clock hours shall be required in the basic science training programs, subdivided as follows:

a) A minimum of 10 clock hours each in the following subjects: anatomy, physiology, biochemistry, pathology, toxicology, and pharmacology.

b) The remainder of the minimum required hours may be taken in any of the subjects mentioned or in other basic sciences related to the specialty of anesthesiology.

Those applicants who qualify must submit acceptable documentary evidence, which may be further investigated at the discretion of this Board, of having pursued this advanced training in colleges, laboratories, clinics, hospitals, or other facilities satisfactory to this Board.

9. He shall provide evidence that he has practiced the specialty of anesthesiology for a period of at least 2 years subsequent to the completion of the required minimum of 3 years of formal training, or its equivalent, prior to being examined.

10. He shall provide evidence that he has administered a minimum of 1,250 anesthetics of a diversified nature.

11. He shall demonstrate his fitness to take the examination in any other manner required by this Board.

12. He shall present detailed case records, acceptable to this Board, of 25 major anesthetic procedures, using an outline furnished by this Board, which correlates the principal details of preoperative examination, the reasons for selection and utilization of the anesthetic agents, operative and postoperative conduct of the anesthesia.

13. He shall submit complete anesthetic case records and a report of pertinent factors where fatalities resulted within 24 hours in all patients anesthetized by the applicant during the 12-month period immediately prior to making application for examination by this Board.

14. He shall submit a list of the anesthetics administered by him for the 12-month period immediately prior to making application, naming the type of operation, the hospital in which the operation was performed, the patient's initials, the hospital case number, the name of the surgeon, the anesthetics administered, and the case termination. This list shall be certified by the administrator of the hospital in which the anesthetics were administered.

15. He shall submit a segregated total of all anesthetics administered in the 3-year period prior to making application, thereby showing the variety of agents and methods of anesthesia.

16. In the case of any applicant who was engaged in the practice of anesthesiology by 1939, or prior thereto, and who graduated prior to 1936, and who meets the general requirements stated in Article IV, Section 1, a), b), c), d), and e) of the Bylaws, and who has maintained appropriate activity in American Osteopathic Association organizational work, specialty societies, hospital staff work, the pursuit of periodic postgraduate work, contributions to professional literature or otherwise has indicated his consistent interest and competence in the field of anesthesiology, the requirements of Article IV, Section 1, f) and h) of the Bylaws may be waived and the applicant may be recommended for certification after passing an appropriate oral and/or clinical examination.

How much enjoyment can your geriatric patients get from life?

OF course, with the help of a good well-balanced diet, your older patients can be healthier and more active. That problem of diet is where Gerber's can be of tremendous assistance to you.

First of all, Gerber's variety is wide enough to allow for your patient's "foibles" about food — some of them built up over years and years. Yet that same variety gives you good prescription selectivity.

To help provide both the known and "unknown" nutritive factors, all of Gerber's Strained and Junior (Chopped) Foods are specially processed to retain natural food values to the maximum possible by modern methods.

Added encouragement to staying with your prescribed regimen—Gerber's "Special Diet Recipes." They offer a tempting range of easy-to-make dishes for many tastes. FREE COPIES of this booklet, with recipes based on Bland, Soft, Mechanically Soft, Liquid and Low-Residue Diets. Just write on your letterhead to Dept. 372-5, Fremont, Mich.

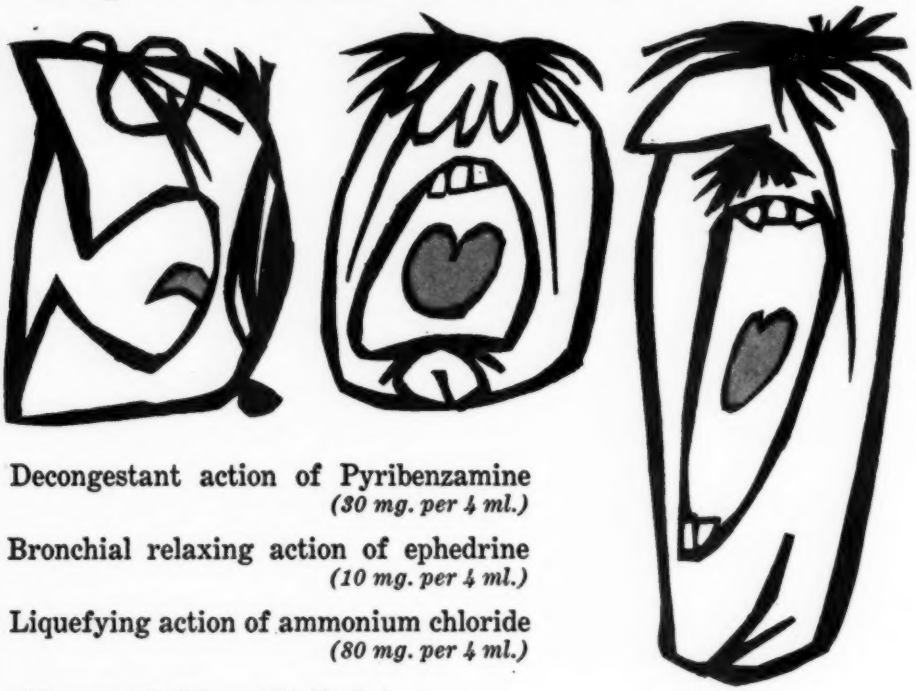
Gerber's BABY FOODS

4 CEREALS • 60 STRAINED & JUNIOR MEATS, VEGETABLES, FRUITS, DESSERTS

coughing patients need

Pyribenzamine®

Expectorant with Ephedrine for



Decongestant action of Pyribenzamine
(30 mg. per 4 ml.)

Bronchial relaxing action of ephedrine
(10 mg. per 4 ml.)

Liquefying action of ammonium chloride
(80 mg. per 4 ml.)

Also available with Codeine
(8 mg. per 4 ml.)

Pyribenzamine® citrate (tripelennamine citrate CIBA)

CIBA
SUMMIT, N. J.

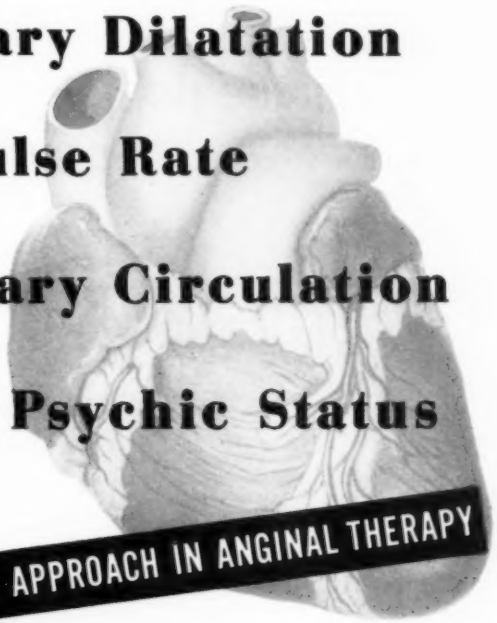
2/208436

Lasting Coronary Dilatation

Lower Pulse Rate

Better Coronary Circulation

Improved Psychic Status



THE NEW, MORE SUCCESSFUL APPROACH IN ANGINAL THERAPY

PENTOXYLON combines the bradycrotic, tranquilizing, stress-relieving effects of Rauwiloid® 1 mg. with the prolonged coronary vasodilating influence of pentaerythritol tetranitrate (PETN) 10 mg.

Reduced heart rate lengthens diastole and leads to better coronary filling and wider stroke volume.

This new approach reduces nitroglycerin need, in many instances obviates it; increases exercise tolerance, reduces anxiety, allays apprehension, and often produces objective improvement demonstrable by electrocardiogram.

Dosage: one to two tablets q.i.d. In bottles of 100 tablets.

PENTOXYLON™

Each tablet contains pentaerythritol tetranitrate (PETN) 10 mg. and Rauwiloid® 1 mg.

Equally indicated in normotensive and hypertensive patients, since Rauwiloid lowers elevated blood pressure but does not affect normal tension.

Riker LABORATORIES, INC., LOS ANGELES 48, CALIF.

relieve pain, headache, fever promptly and safely

APAMIDE®

(N-acetyl-p-aminophenol, Ames, 0.3 Gm.)

direct-acting analgesic-antipyretic...

no toxic by-products...

APAMIDE-VES

TRADEMARK

(Buffered N-acetyl-p-aminophenol, Ames, 1 Gm.)

effervescing analgesic-antipyretic...

speeds relief...assures fluid intake

APROMAL®

(acetylcarbromal and N-acetyl-p-aminophenol, Ames, 0.15 Gm. ea.)

sedative-analgesic-antipyretic...

calms patients and relieves pain

AMES

COMPANY, INC • ELKHART, INDIANA

Ames Company of Canada, Ltd., Toronto 59554

We Cannot Tell a Lie about RIASOL IN PSORIASIS

Like the famous boy who chopped down the cherry tree, we confess the whole truth about RIASOL.

In 24% of a series of patients with psoriasis RIASOL failed to improve the skin condition. Yet other treatments had failed in 100% of this same series.

In 19% of the cases treated with RIASOL the disease recurred. Yet it was quickly controlled in most cases by resuming RIASOL treatment.

It took an average of 8 weeks before the skin lesions cleared under treatment with RIASOL. Yet the average duration of psoriasis in this series was 8 years.

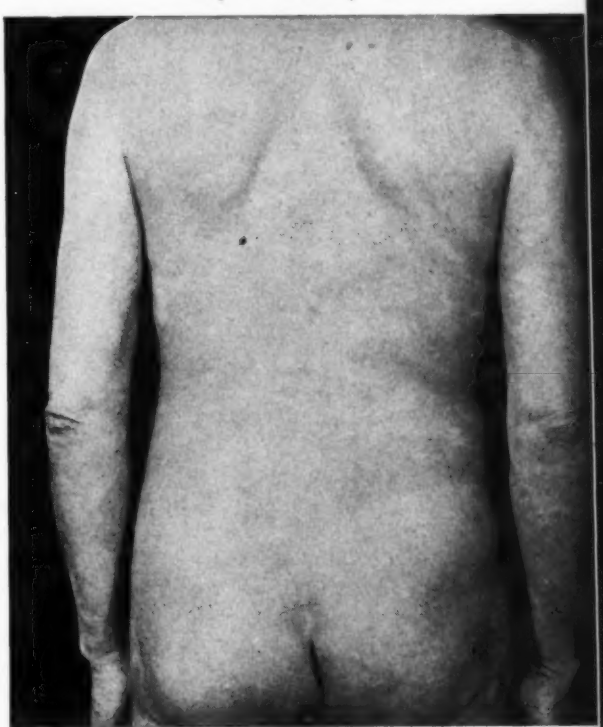
RIASOL is not infallible in the treatment of psoriasis. But the chances are all in your patient's favor.

RIASOL contains 0.45% mercury chemically combined with soaps, 0.5% phenol and 0.75% cresol in a washable, non-staining, odorless vehicle.

Apply daily after a mild soap bath and thorough drying. A thin, invisible, economical film suffices. No bandages required. After one week, adjust to patient's progress.

RIASOL is supplied in 4 and 8 fld. oz. bottles at pharmacies or direct.

Before Use of Riasol



After Use of Riasol

MAIL COUPON TODAY— TEST RIASOL YOURSELF

SHIELD LABORATORIES JAOA-2-55

12850 Mansfield Ave., Detroit 27, Mich.

Please send me professional literature and generous clinical package of RIASOL.

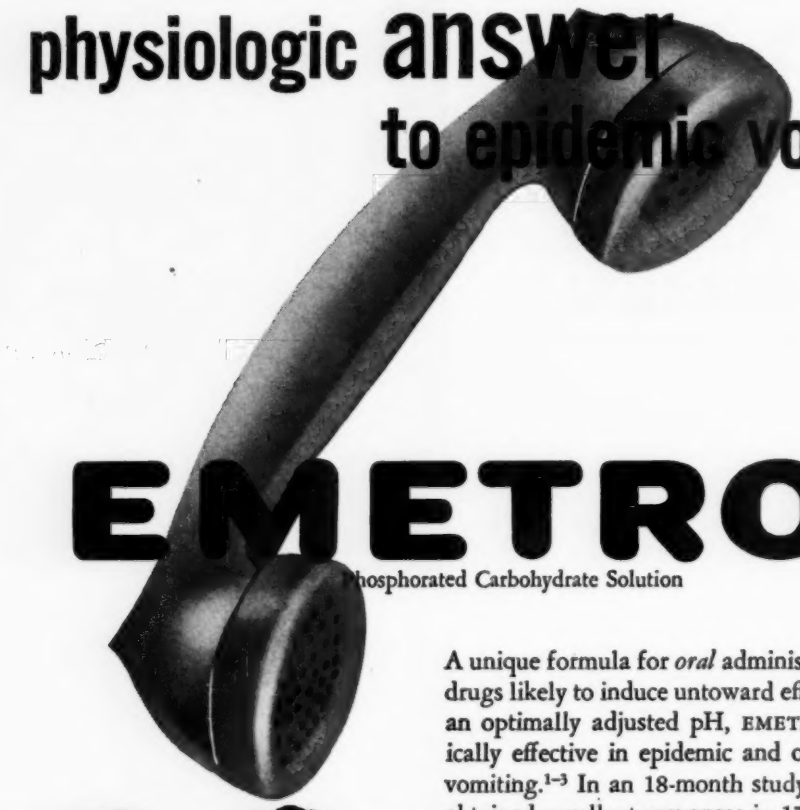
..... D.O. .....Street

City..... Zone..... State.....

Druggist..... Address.....

RIASOL for PSORIASIS

physiologic answer to epidemic vomiting



EMETROL®

Phosphorated Carbohydrate Solution

A unique formula for *oral* administration . . . containing no drugs likely to induce untoward effects . . . and stabilized at an optimally adjusted pH, EMETROL has proved dramatically effective in epidemic and other types of functional vomiting.[1-3] In an 18-month study, Bradley and associates obtained excellent responses in 172 children, often with a single dose of 1 to 3 teaspoonfuls. EMETROL is easy and pleasant to take, safe for all age groups.

IMPORTANT: EMETROL must always be taken *undiluted.* No fluids should be allowed for at least 15 minutes after each dose.

DOSAGE: For infants and children, 1 or 2 teaspoonfuls every 15 minutes until vomiting stops. For adults, 1 or 2 tablespoonfuls.

SUPPLIED: In bottles of 3 fl.oz. and 16 fl.oz., through all pharmacies.

In nausea of pregnancy, EMETROL has produced favorable response in 3 out of every 4 cases, usually within 24-48 hours.[2] Recommended as "free of annoying side effects . . . a safe and physiologic agent . . ."

1. Bradley, J. E., et al.: J. Pediat. 38:41, 1951. 2. Crunden, A. B., Jr., and Davis, W. A.: Am. J. Obst. & Gynec. 65:311, 1953. 3. Tebrock, H. E., and Fisher, M. M.: M. Times 82:271, 1954.

Literature and sample on request

KINNEY & COMPANY, INC.
COLUMBUS, INDIANA

first... for fast relief
INJECT
PHYATROMINE-H
U.S. Pat. 2,678,899 Canad. Pat. 503,585 1954

relieves crippling skeletal muscle spasm without affecting smooth muscle. Quickly increases range of motion and decreases pain/spasm.

Each cc. contains: Physostigmine salicylate, 0.6 mg., and l-hyoscyamine hydrobromide, 0.3 mg.

SUPPLIED: 1-cc. ampuls, boxes of 25; 10- and 30-cc. multiple dose vials. See package circular for dosage.

IN CRIPPLING MUSCLE SPASM
K·U inject-oral therapy

as in all types of arthritis, myositis, bursitis, fibrositis, low-back pain, sprains and strains of muscles and joints.

then... for sustained comfort
PRESCRIBE
SALIMEPH-C tablets
TRADEMARK

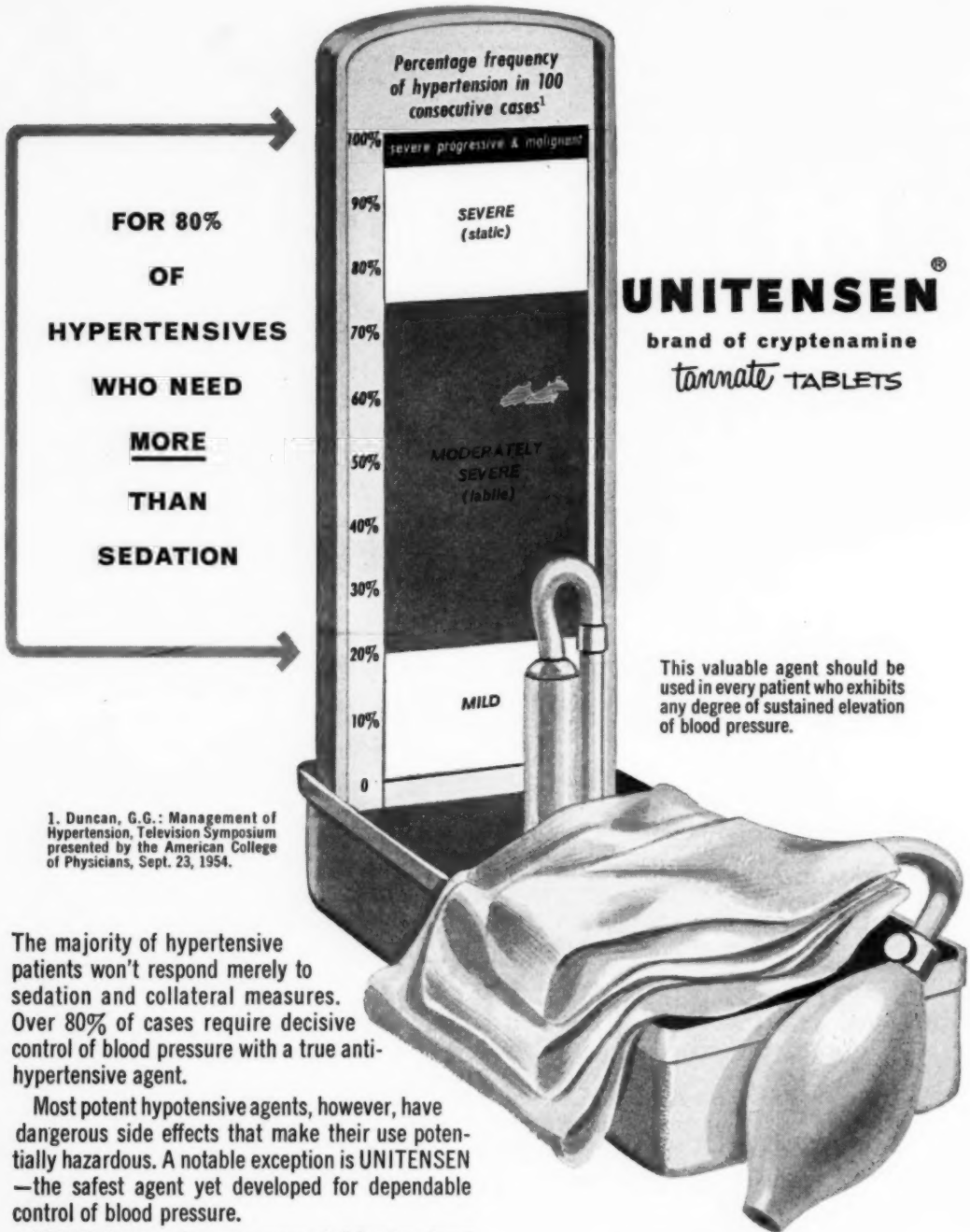
relieves the pain that causes spasm, and relaxes the spasm that causes pain... without affecting consciousness or normal muscular movements, and without gastric irritation.

Each tablet contains: Salicylamide, 250 mg.; mephenesin, 250 mg., and ascorbic acid, 15 mg.

DOSAGE: 2 or 3 tablets q.i.d.

SUPPLIED: Bottles of 100, 500 and 1000 tablets.

Ethical Pharmaceuticals Since 1894
KREMERS-URBAN COMPANY Laboratories in Milwaukee



FOR 80% OF HYPERTENSIVES WHO NEED MORE THAN SEDATION

UNITENSEN®

brand of cryptenamine

tannate TABLETS

This valuable agent should be used in every patient who exhibits any degree of sustained elevation of blood pressure.

1. Duncan, G.G.: Management of Hypertension, Television Symposium presented by the American College of Physicians, Sept. 23, 1954.

The majority of hypertensive patients won't respond merely to sedation and collateral measures. Over 80% of cases require decisive control of blood pressure with a true antihypertensive agent.

Most potent hypotensive agents, however, have dangerous side effects that make their use potentially hazardous. A notable exception is UNITENSEN—the safest agent yet developed for dependable control of blood pressure.

With Unitensen, the ever present risk of postural hypotension and collapse associated with ganglionic blocking agents does not exist. Unitensen is assayed twice—for hypotensive and emetic action—to assure a workable range between hypotensive response and emetic effect.

Each white, scored tablet contains:

Cryptenamine* . . . . . 2 mg.†
(as the tannate salt)

Bottles of 50, 100, 500 and 1000.

*ester alkaloids of Veratrum viride obtained by an exclusive Irwin-Neisler nonaqueous extraction process.
†equivalent to 260 Carotid Sinus Reflex Units

IRWIN, NEISLER & COMPANY • DECATUR, ILLINOIS • TORONTO I, ONTARIO

Conventions and Meetings

Announcements

American Osteopathic Association, Fifty-Ninth Annual Convention, College of Osteopathic Physicians and Surgeons and Biltmore and Statler Hotels: Convention Instruction Courses, July 5-16; Convention Teaching Sessions, July 18-22. Program Chairman, W. Donald Baker, 3450 W. 43rd St., Los Angeles 8.

American College of Osteopathic Surgeons, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Administrative Secretary, Mrs. E. F. Martin, Box 474, Coral Gables 34, Fla. Program Chairman, Arthur M. Flack, 135 S. 17th St., Philadelphia 3.

American Osteopathic Academy of Orthopedics, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Secretary, J. Paul Leonard, 2673 W. Grand Blvd., Detroit 8.

American Osteopathic College of Anesthesiologists, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Secretary, Crawford M. Esterline, Box 155, Kirksville, Mo. Program Chairman, Harry J. Petri, Jr., 5 Deering St., Portland 3, Maine.

American Osteopathic College of Radiology, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Secretary, F. A. Turfler, Jr., South Bend Osteopathic Hospital, 118 S. William St., South Bend 2, Indiana.

American Osteopathic Hospital Association, annual meeting, Hotel Statler, Washington, D. C., October 30-November 3. Executive Secretary, Mr. R. P. Chapman, 1013 Kahl Bldg., 326 W. Third St., Davenport, Iowa.

American Osteopathic Society of Proctology, annual clinical assembly, Bellevue-Stratford Hotel, Philadelphia, April 14-16. Secretary, Carl S. Stillman, Jr., 3523 Fifth Ave., San Diego 3, Calif. Program Chairman, Joseph W. Kenney, 718 Guaranty Bldg., Indianapolis 4.

Arizona, annual meeting, Paradise Inn. Phoenix, April 22-24. Secretary, H. E. Allshouse, 2243 N. 12th St., Phoenix 22.

Arkansas, annual meeting, Albert Pike Hotel, Little Rock, May 12-13. Secretary, R. M. Packard, 409 Citizens Bank Bldg., Jonesboro. Program Chairman, L. E. O'Keefe, First State Bank Bldg., Springdale.

Canada, annual meeting, Mount Royal Hotel, Montreal, October 29-31. Secretary, Miss Joyce S. Currie, 609 Medical Arts Bldg., Montreal 25. Program Chairman, A. E. Wilkinson, 616 Medical Arts Bldg., Montreal 25.

Eastern Osteopathic Association, annual meeting, Hotel Statler, New York City, April 2-3. Secretary, Frank B. Tompkins, Baltimore Life Bldg., Baltimore 1. Program Chairman, Chester D. Losee, 212 Prospect St., Westfield, N. J.

Florida, annual meeting, Orange Court Hotel, Orlando, April 14-16. Secretary, Dominic Raffa, 5009 Central Ave., Tampa 3. Program Chairman, Charles C. Tindall, 119 Dakin Ave., Kissimmee.

Georgia, annual meeting, Bon Air Hotel, Augusta, April 29-30. Secretary, Walter B. Elliott, Jr., 702 Mortgage Guarantee Bldg., Atlanta 3. Program Chairman, M. Lillian Bell, 2075 Ridgewood Drive, N. E., Atlanta 6.

Illinois, annual meeting, Faust Hotel, Rockford, April 29-May 1. Executive Secretary, Mr. Douglas Durkin, 5200 S. Ellis Ave., Chicago 15. Program Chairman, E. C. Andrews, Ottawa Arthritis Hospital and Diagnostic Clinic, Ottawa.

Indiana, annual meeting, French Lick Springs Hotel, French Lick Springs, May 14-17. Secretary, Arabelle B. Wolf, 809-13 Odd Fellows Bldg., Indianapolis 4. Program Chairman, Paul van B. Allen, 1500 N. Delaware St., Indianapolis 2.

Kansas, annual meeting, Allis Hotel, Wichita, April 23-27. Executive Secretary, Mr. Lloyd L. Hall, Room 612, National Bank of Topeka Bldg., Topeka. Program Chairman, R. G. Waddill, 2914 E. Central, Wichita 6.

Maine, annual meeting, Samoset Hotel, Rockland, June 16-18. Midyear meet-

. . . no two hypertensives are alike

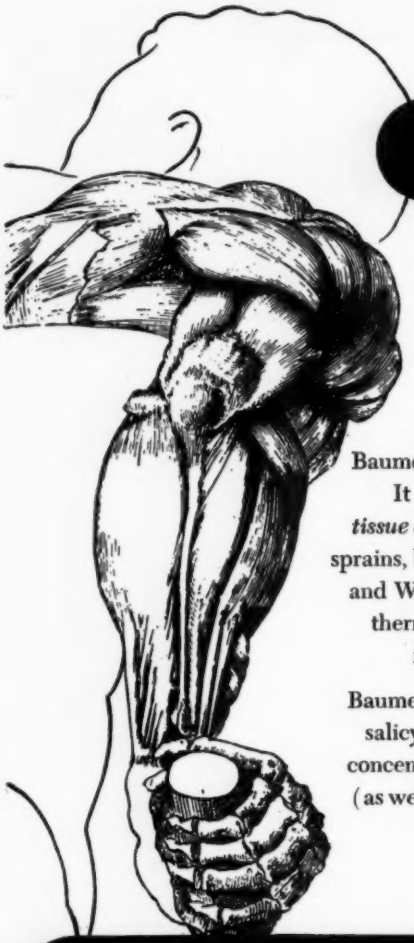
Joe Smith is on Crystoserpine
Diagnosis: Emotionally disturbed; labile hypertension Grade I

J. S., greatly agitated individual, is now on Crystoserpine (crystalline reserpine-Dorsey), 1 tablet of 0.25 mg. q. i. d. because he is emotionally disturbed, nervous, ambitious but often frustrated. His work means unrelenting pressure, and as a result he developed a mild, labile hypertension (Grade 1). After three weeks of therapy he is a calm, tranquil individual. His blood pressure and slightly elevated pulse rate are down to normal. At no time did Crystoserpine cause postural hypotension or toxic effects. Now his family can "live" with him.

Crystoserpine* is a DORSEY preparation. Supplied in bottles of 100, 500, and 1,000 tablets.

Smith-Dorsey • Lincoln, Nebraska • A Division of The Wander Company
*TRADE MARK

Joe Smith does best on Crystoserpine—for no two hypertensives are alike.



going deep

The "hyperkinemic" activity of Baume Bengué goes beneficially deep. It enhances blood flow *through the tissue area* in arthritis, myositis, muscle sprains, bursitis and arthralgia. As Lange and Weiner[1] determined by the use of thermo-needles, hyperkinemic effect may extend to a depth of 2.5 cm.

Baume Bengué also promotes systemic salicylate action. It provides the high concentration of 19.7% methyl salicylate (as well as 14.4% menthol) in a specially prepared lanolin base to foster percutaneous absorption.

Baume Bengué
ANALGESIQUE

1. Lange, K., and Weiner, D.: J. Invest. Dermat. 12:263 (May) 1949.

Available in both **regular** *and* **mild** *strengths*

Thos. Leeming & Co. Inc. 155 East 44th Street, New York 17, N.Y.

ing, Eastland Hotel, Portland, December 1-3. Secretary, Roswell P. Bates, 72 Main St., Orono.

Michigan, annual meeting, Civic Auditorium, Grand Rapids, October 3-5. Executive Secretary, Mr. Harve Lamont Smith, 81 Glendale, Highland Park 3. Program Chairman, Harold W. Laidlaw, McCoy-Laidlaw Clinic, Bad Axe.

Minnesota, annual meeting, Lowry Hotel, St. Paul, May 6-7. Secretary, E. R. Komarek, 301 Granite Exchange Bldg., St. Cloud. Program Cochairmen, M. Sidney Hedeen, 1593 University Ave., St. Paul 4, and Brian F. Doherty, 925 New York Bldg., St. Paul 1.

National Osteopathic Child's Health Conference and Clinic, Municipal Auditorium, Kansas City, Mo., April 4-6. Program Chairman, John C. Taylor, 3504 Troost Ave., Kansas City 3, Mo.

New Jersey, annual meeting, Ritz Carlton Hotel, Atlantic City, March 12-13. Executive Secretary, Mr. I. J. Tecker, 21 Hancock St., Riverside. Program Chairman, Mortimer J. Sullivan, 21 N. Mountain Ave., Montclair.

New Mexico, annual meeting, Alvarado Hotel, Albuquerque, May 19-21. Secretary, Robert E. Smith, 205 N. First St., Lovington.

New York, midyear meeting, Hotel Powers, Rochester, February 26-27. Secretary, Robert E. Cole, 417 S. Main St., Geneva. Program Chairman, Myron C. Beal, 17 Vick Park A, Rochester 7. Annual meeting, Hotel Statler, New York City, October 14-15. Program Chairman, Walter Streicker, 7 Plaza St., Brooklyn 17.

Northwest Osteopathic Convention, annual meeting, Portland, Ore., June 13-16. Program Chairman, Russell F. Kenaga, Sr., 2265 W. Burnside St., Portland 5.

Ohio, annual meeting, Neil House, Columbus, May 2-4. Executive Secretary, Mr. William S. Konold, 50 E. Broad St., Columbus 15. Program Chairman, Robert H. Gibson, 112 W. Third Ave., Columbus 1.

Ontario, annual meeting, Empress Hotel, Peterborough, May 6-7. Secretary, A. V. DeJardine, 205 Yonge St., Toronto 1. Program Chairman, Reginald G. Martin, 124 Hunter St., W., Peterborough.

Oregon: See Northwest Osteopathic Convention.

Osteopathic College of Ophthalmology and Otorhinolaryngology, annual meeting, Continental Hotel, Kansas City, Mo., July 25-27. Executive Secretary, Mr. William S. Konold, 50 E. Broad St., Columbus 15, Ohio. Program Chairman, H. Mahlon Gehman, 1818 Pine St., Philadelphia 3.

Rhode Island, annual meeting, Warwick Country Club, Warwick, March 2-3. Secretary, J. Weston Abar, 824 Broad St., Providence 7. Program Chairman, Herbert L. Adams, 922 Reservoir Ave., Cranston 10.

South Dakota, annual meeting, Sylvan Lake Hotel, Black Hills, June 5-7. Secretary, E. W. Hewlett, 417 W. 27th St., Sioux Falls. Program Chairman, Laurence S. Betts, Box 205, Huron.

Tennessee, annual meeting, Maxwell House Hotel, Nashville, March 21-23. Secretary, J. M. Moore, Jr., 200 High St., Trenton. Program Chairman, Fred L. Mitchell, 516 James Bldg., Chattanooga 2.

Texas, annual meeting, Rice Hotel, Houston, May 12-14. Executive Secretary, Phil R. Russell, 512 Bailey St., Fort Worth 7. Program Chairman, J. Natcher Stewart, Stevens Park Osteopathic Hospital, 1141 N. Hampton Road, Dallas 11.

Utah and Wyoming, joint annual meeting, Logan, Utah, June 26-27. Secretary, Utah Society of Osteopathic Physicians and Surgeons, Alice E. Houghton, 600 Zion's Savings Bank Bldg., Salt Lake City 1. Secretary, Wyoming Association of Osteopathic Physicians and Surgeons, G. A. Roulston, 2823 Central St., Cheyenne.

Virginia, annual meeting, The Lodge, Williamsburg, May 27-28. Acting Secretary, Harold A. Blood, 228 N. Co-

lumbus St., Alexandria.

West Virginia, annual meeting, McClure Hotel, Wheeling, June 4-7. Secretary, Guy E. Morris, 542 Empire Bank Bldg., Clarksburg. Program Chairman, Joseph B. C. Bartram, Gilmer Clinic, Post Office Bldg., Glenville.

Wisconsin, annual meeting, Hotel Pfister, Milwaukee, April 18-20. Secretary, Edwin J. Elton, 1518 N. 70th St., Wauwatosa 13. Program Chairman, Paul A. Allen, 6 S. Mill St., Waupun.

Wyoming: See Utah.

State and National Boards

ARIZONA

Those interested in professional examinations should contact Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination in Medicine and Surgery, 2747 East McDowell Road, Phoenix.

L. A. Nowlin, Phoenix, has been appointed to the Board.

Basic science examinations March 15 at the University of Arizona, Tucson. Applications must be filed 2 weeks prior to the examination. Address Herbert D. Rhodes, Ph.D., secretary, Basic Science Board, University of Arizona, Tucson.

COLORADO

Basic science examinations March 2-3 in the Lecture Room, second floor, Y.M.C.A. Bldg., E. 16th Ave. and Lincoln St., Denver. Applications must be filed by February 16. Address Esther B. Starks, D.O., secretary, Basic Science Board, 1459 Ogden St., Denver 18.

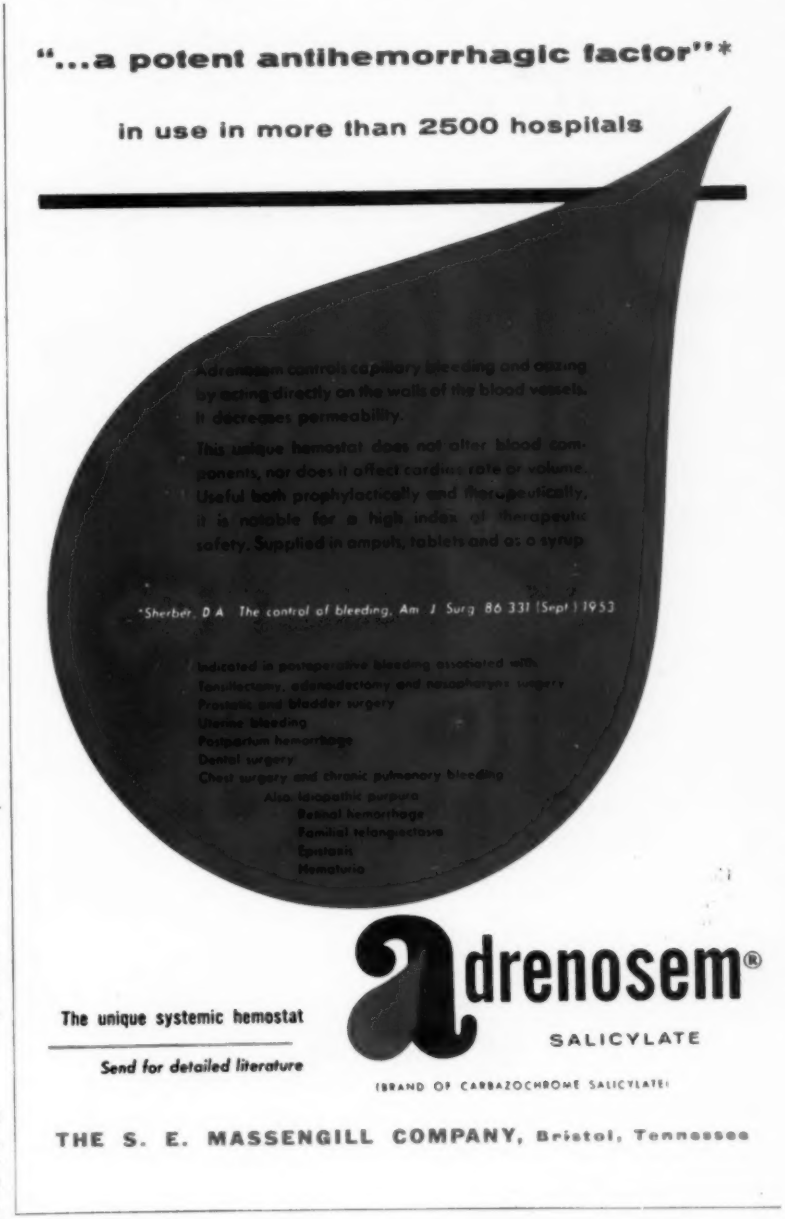
DISTRICT OF COLUMBIA

Professional examinations May 9-10. Address Mr. Paul Foley, Deputy Director, Department of Occupations and Professions, 1740 Massachusetts Ave., N.W., Washington 6.

Basic science examinations in April. Address Mr. Foley.

FLORIDA

The officers of the Board of Osteopathic Medical Examiners are: Chairman, Edmund T. Flynn, Tallahassee; vice chairman, Louis J. Larmoyeux, Jacksonville; secretary-treasurer, R. Philip Coker, Panama City.


"...a potent antihemorrhagic factor"*
in use in more than 2500 hospitals
Adrenosem controls capillary bleeding and oozing by acting directly on the walls of the blood vessels. It decreases permeability.
This unique hemostat does not alter blood components, nor does it affect cardiac rate or volume. Useful both prophylactically and therapeutically, it is notable for a high index of therapeutic safety. Supplied in ampuls, tablets and as a syrup.
*Sherbet, D A The control of bleeding, Am J Surg 86 331 (Sept.) 1953
Indicated in postoperative bleeding associated with:
Tonsillectomy, adenoidectomy and nasopharynx surgery
Prostatic and bladder surgery
Uterine bleeding
Postpartum hemorrhage
Dental surgery
Chest surgery and chronic pulmonary bleeding
Also: Idiopathic purpura
Retinal hemorrhage
Familial telangiectasia
Epistaxis
Hematuria
adrenosem®
SALICYLATE
(BRAND OF CARBAZOCHROME SALICYLATE)
The unique systemic hemostat
Send for detailed literature
THE S. E. MASSENGILL COMPANY, Bristol, Tennessee

ILLINOIS

Examinations in April in Chicago. Address Mr. Frederic B. Selcke, Superintendent of Registration, Department of Registration and Education, State House, Springfield.

IOWA

Basic science examinations April 12 at the Capitol Bldg., Des Moines. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.

KANSAS

Examinations February 17-19 at Topeka. Address Forrest H. Kendall, D.O., secretary, Board of Osteopathic Examination and Registration, 420½ Pennsylvania, Holton.

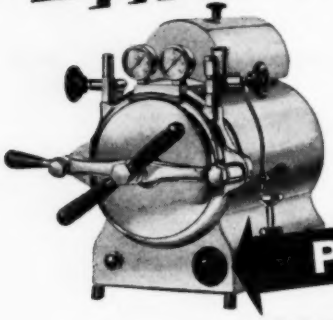
MICHIGAN

Basic science examinations May 14-15 at Detroit and Ann Arbor. Address Mrs. Anne Baker, secretary, Board of Examiners in the Basic Sciences, 116 Mason Bldg., Lansing.

MINNESOTA

Basic science examinations April 5-6 at the University of Minnesota, Minneapolis. Applications must be filed by March 10. Address Raymond N. Bieter, M.D., secretary, 105 Millard Hall, University of Minnesota, Minneapolis 14.

HIGH SPEED INSTRUMENT STERILIZATION *at the twist of a* PELTON DIAL



COMPLETE AUTOCLAVING and STERILIZING *in less time than simple boiling*

With any of the three Pelton Autoclaves . . . FL-2 (6″ x 12″), HP-2 (8″ x 16″ — illustrated above) and now the new LV-2 (12″x 22″) . . . you are ready for fast sterilizing of instruments in any emergency. After the autoclave is in operation, you can sterilize uncovered instruments in only three minutes by turning the pressure dial to 27 lbs. (267° F.). Set at normal 250° F., wrapped instruments are safely sterilized in 15 minutes.

IDEAL STAND-BY EQUIPMENT FOR EVERY HOSPITAL and CLINIC

Generating its own steam and storing it for immediate use, a Pelton Autoclave is always ready to sterilize an urgently needed instrument, or one that has been dropped, in a matter of a few minutes.

Call your Pelton dealer or write today for literature.

Professional Equipment Since 1900

PELTON

THE PELTON & CRANE CO. • DETROIT 2, MICHIGAN

MONTANA

Examinations March 1. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

NEBRASKA

Basic science examinations May 3-4. Address Mr. Husted K. Watson, Director, Bureau of Examining Boards, Department of Health, State Capitol Bldg., Lincoln 9.

NEVADA

Basic science examinations April 5. Address Donald G. Clooney, Ph.D., secretary, Board of Examiners in the Basic Sciences, Box 9005, University Station, Reno.

NEW HAMPSHIRE

Examinations March 9-10 at Concord. Address John S. Wheeler, M.D., secretary, Board of Registration in Medicine, State House, Concord.

NEW MEXICO

Basic science examinations April 17. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, P. O. Box 1522, Santa Fe.

NEW YORK

Examinations February 15-18. Applications must be filed not less than 30 days prior to examination. Address Dr. John W. Paige, Chief, Bureau of Professional Examinations and Registrations, 23 S. Pearl St., Albany 7.

OKLAHOMA

Basic science examinations April 7-8 at the University of Oklahoma School of Medicine. Applications must be filed 10 days prior to examination. Address Secretary of State, State Capitol Bldg., Oklahoma City.

OREGON

Basic science examinations March 5 in Rooms 207-208, Portland State Extension Center Bldg., 1620 S.W. Park St., Portland. Applications must be filed by February 16. Address Mr. John R. Richards, secretary, Board of Higher Education, Eugene.

RHODE ISLAND

Professional examinations April 7-8. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

Basic science examinations in Room 366, State Office Bldg., Providence, May 18. Applications must be filed approximately 20 days prior to examination. Address Mr. Casey.

TENNESSEE

Examinations in February and July at Nashville. Applications must be filed 15 days prior to examination. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

WISCONSIN

Basic science examinations March 12 at Hotel Loraine, Madison. Applications must be filed by March 4. Address Prof. William H. Barber, secretary, Board of Examiners in the Basic Sciences, 621 Ransom Ave., Ripon.

REREGISTRATION OF OSTEOPATHIC LICENSES

Before March 1—Colorado, $2.00 if legal resident; $10.00 if not legal resident. Address Samuel H. Brown, M.D., executive secretary, Board of Medical Examiners, 831 Republic Bldg., Denver 2.

April 1—Montana, $2.00 for those in active practice; $1.00 for those inactive. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

April 1—Wyoming, $2.50. Address Franklin D. Yoder, M.D., secretary, Board of Medical Examiners, New State Office Bldg., Cheyenne.

May 1—Iowa, $1.00. Address Mr. Dwight S. James, assistant secretary, Board of Osteopathic Examiners, 200 Walnut Bldg., Des Moines 9.

May 1—Washington, $2.00. Address Mr. Edward C. Dohm, secretary, Professional Division, Department of Licenses, Olympia.

EXAMINATION BY NATIONAL BOARD

The National Board of Examiners for Osteopathic Physicians and Surgeons conducts Parts I and II of its examination on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary or the dean of the college, and the completed application blank, together with a passport photograph and check for the parts to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

Examinations in Part I consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including parasitology and immunology.

Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties; obstetrics and gynecology; pediatrics; neurology and psychiatry; public health, including hygiene, medical jurisprudence; osteopathic principles, therapeutics, including pharmacology and materia medica.

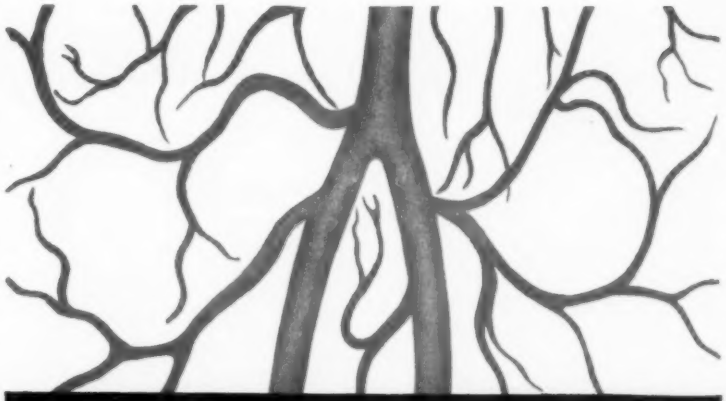
Part III is an oral and practical examination given under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles, therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

Part III examinations will be given at the College of Osteopathic Physicians and Surgeons, Los Angeles, March 5-6, at the Chicago College of Osteopathy, Chicago, April 3-4, and in the Spring at Philadelphia and Kirksville, Mo.

These are oral examinations which the candidate may take after having satisfactorily completed the first 6 months of a 1-year internship in a hospital approved by the American Osteopathic Association for intern training. Part III is given annually at the above-named colleges.

Eligibility requirements are as follows: Part I, satisfactory completion of the first 2 years in an approved school of osteopathy; Part II, satisfactory completion of Part I and of the first two quarters or trimesters of the senior year in an approved osteopathic college; Part III, satisfactory completion of Part II and at least 6 months of a 1-year internship approved by the American Osteopathic Association. The internship requirement does not apply to candidates who took Part I prior to July, 1950.

Application must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.



"...Unique blood-clotting faculty, acting promptly...will often obviate the use of transfusion....Preoperatively tends to reduce blood loss and to facilitate surgical procedures....Over an eleven-year period no untoward effects...."*

KOAGAMIN is a parenteral hemostat containing oxalic and malonic acids in aqueous solution. Supplied in 10-cc. diaphragm-stoppered vials.

*Joseph, M.: Am. J. Surg. 87:905, 1954.

Chatham

CHATHAM PHARMACEUTICALS, INC.
Newark 2, New Jersey

04484

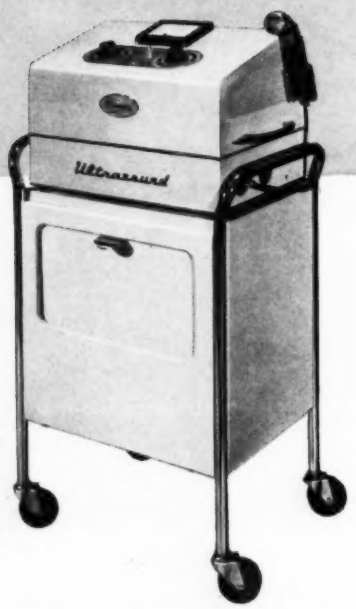
NATIONWIDE INCREASE OF FAMILIES*

The great willingness of the present generation to take on the responsibilities of family life is reflected by the record number of persons who married after World War II and by the continuing boom in births. During the 1940's, the number of families in the United States increased by 6.1 million, or by 19 percent, bringing the total to about 38.3 million in April 1950. In the decade between 1930 and 1940, the increase was only 4.2 million, or 15 percent. Moreover, the number of families has continued to grow since 1950, and is now at an all-time high.

Every section of the United States had more families in 1950 than in 1940. The gain varied markedly, however, from State to State. Practically no increase—only 1 percent—occurred in three South Central States, namely, Mississippi, Arkansas, and Oklahoma. Relatively small gains—less than 10 percent—were experienced by Vermont and Kentucky, and by five of the West North Central States. At the other extreme, six States recorded gains far in excess of the national average. In Arizona, California, Florida, and Nevada the number of families increased by more than one half,

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, November, 1954.

Announcing...



THE Burdick

ULTRASONIC
Therapy Unit

Years of Burdick research and experience have resulted in the development of this outstanding unit.

From controls to applicator, the UT-1 Ultrasonic unit is constructed to provide you the maximum in therapeutic efficiency, durability and safety — typical features of all Burdick equipment.

Write for complete descriptive literature.

THE BURDICK CORPORATION
MILTON, WISCONSIN

and in Oregon and Washington by about two fifths. It is noteworthy that these States led the country in population growth, and that all but one are located in the West.

The marked geographic variations in increase of families were due largely to the interstate movement of population. Each of the States with a small increase of families during the decade from 1940 to 1950 sustained a loss of population by migration, whereas the States with a marked increase of families were those which attracted a relatively large number of migrants. The record influx of population into California was the major factor in that State's gain of one million families. As a result, California has passed Pennsylvania and Illinois in number of families, and is now outranked only by New York.

About 138 million persons, or more than nine tenths of the population in the United States, were living in family units in 1950. Family size was smallest in the District of Columbia and along the Pacific Coast, where it averaged about 3.3 persons per family. On the other hand, States with substantial farm populations, particularly those in the South, tended to have the largest number of persons per family. The Carolinas and Mississippi, for example, had somewhat more than four persons per family.

In view of the record number of births in recent years, it may come as a surprise to many readers that the average number of persons in an American family has decreased. For the country as a whole, the average number per family declined from 3.77 in 1940 to 3.60 in 1950. A number of factors account for this paradox. Foremost, of course, was the marriage boom which reduced the number of persons in existing families and at the same time added many new families with a small number of persons. Then, too, the increase in the size of our Armed Forces since 1940 has withdrawn millions of our men from their homes. Finally, favorable economic conditions and the large volume of housing construction enabled thousands of married couples and other groups of related persons to establish independent households. As a matter of fact, the increase in households—23 percent for the country as a whole—was greater than the rate of increase for families in every area of the country outside of the West, and in that region the rates of growth for households and families were practically on a par.

FACTORS IN MENTAL RETARDATION*

George A. Jervis, M.D., Ph.D.
Director of Clinical Laboratories,
Letchworth Village
New York State Department of Mental Hygiene

Various sciences have contributed to our present concept of mental deficiency. For a long time sociologists have observed that there are individuals who, since childhood, have been socially incompetent and incapable of adequate self-support. Psychologists, coming later, have noted that this social incompetence is often associated with defective intellectual development. They have discovered ways of measuring the degree of intellectual deficit and of establishing certain correlations between intellectual endowment and social attainments. Then as medical science advanced physicians became increasingly aware that some diseases occurring during fetal life or in infancy may result in lesions of the brain with consequent mental defect. Finally, with the advent of the science of human genetics the relevance of genetic factors in determining deviations of intelligence emerged.

Mental deficiency may be defined as a condition of arrest or incomplete mental development existing before adolescence, caused by disease or genetic constitution and resulting in social incompetence. This definition includes both the sociological concept which stresses the social inadequacy of the defective, and the psychological concept which is considered in the term "arrested" or "incomplete" mental development. The biological viewpoint is embodied in the mention of genetic factors and diseases.

Intellectual impairment developing after adolescence is not usually known as mental deficiency but as dementia, a cus-

*Reprinted from *Children*, November-December, 1954.

tomary differentiation for more than a century in both legal and medical thinking, in spite of its dubious validity.

Thus defined, mental deficiency is not a single condition, but a symptom common to diverse conditions of disparate etiologies and of various manifestations.

In the recognition of mental deficiency, the results of psychological examination play the leading role. The mental age (MA) is determined by psychometric tests and the intelligence quotient (IQ) calculated as the rapport of the mental age to the chronological age (CA): $IQ=\frac{MA}{CA}100$. Other factors besides intelligence quotient are taken into consideration, such as educational attainment, emotional reactions, general behavior, and social adjustment. The information from both familial and personal history is carefully evaluated. Finally, a complete medical examination is performed, using modern techniques of clinical and laboratory medicine. It is upon the evidence thus collected that the diagnosis is made.

Considerable difficulty is often experienced in diagnosing the borderline cases between "subnormality" and mental deficiency. The criterion of social adjustment is decisive in these instances.

INCIDENCE AND CLASSIFICATION

In estimating the incidence of mental deficiency, a great deal depends upon the criteria of diagnosis used in the assessment of defective individuals. For instance, if the criterion of social incompetence is adhered to, the incidence will be higher in a strongly competitive urban environment than in rural communities. If a purely psychological criterion is adopted, the test used and the arbitrary point of demarcation between the defective and the nondefective individual will determine to a large extent percentage figures. If one accepts an IQ of 75 instead of one of 70 as the lower limit for the nondefective, the percentage of defective population will be over twice as large. Estimates based on institutional censuses are obviously inadequate and always too low, since only a fraction of the mentally defective population is institutionalized. Those based on large-group testing of school children have their limitations and are perhaps too high. Accurate surveys using modern techniques of securing data and uniform criteria of evaluating intellectual and social development have been few in number and limited in extension.

On the basis of scattered and incomplete data collected from many sources, it may be assumed that the incidence of mental deficiency in the general population is around 1 percent, using IQ below 70 as the criterion. This figure yields a total of 1,500,000 mental defectives in the United States.

Defectives are usually classified into three groups—idiots, imbeciles, and morons, but the corresponding terms of low-grade, medium-grade, and high-grade defective are to be preferred. Defined in sociological terms and in the language of the English Mental Deficiency Act (1927), idiots are persons whose mental defectiveness is of such degree that they are unable to guard themselves against ordinary physical danger. Imbeciles are persons whose mental defectiveness, though less extreme than in idiots, still prevents them from managing themselves or their affairs, or, in the case of children, of being taught to do so. Morons are persons whose mental defectiveness, though not amounting to imbecility, is yet so pronounced that they require care, supervision, and control for their own protection or for the protection of others, or, in the case of children, appear to be permanently incapable of receiving proper benefit from instruction in ordinary schools.

In more precise psychological terms, an idiot is a person having a mental age of less than 3 years, or, if a child, an intelligence quotient of less than 20. An imbecile is a person having a mental age of 3 to 7 years, inclusive, or, if a child, an intelligence quotient from 20 to 49, inclusive. A moron is a person having a mental age of 8 to 11 or 12 years, or, if a child, an intelligence quotient from 50 to 70 (or 75).

Although of considerable value in dealing with practical problems of defectives, both sociological and psychological classifications present limitations, being purely descriptive in character. More comprehensive are medical classifications which follow mainly etiological criteria, grouping patients according to the cause of the defect. While this type of classifi-

...no two hypertensives are alike

Julia Miller does well on Rautensin
Diagnosis: Hypertension Grade II, tachycardia

J. M., active clubwoman, 55, has a moderately high blood pressure and a very rapid heart rate which have been considerably reduced after 35 days on Rautensin (purified Rauwolfia alkaloids—the alseroxylon fraction) on a schedule of 2 tablets (4 mg.) daily, taken at one time before retiring. Now Julia is much calmer and happier than all last year. Later on she will probably do well on a 1 tablet (2 mg.) daily maintenance dose. No postural hypotension and only minor side effects (stuffy nose) have been observed. Rautensin* is a DORSEY preparation. Supplied in bottles of 100, 500, and 1,000 tablets.

Smith-Dorsey • Lincoln, Nebraska • A Division of The Wander Company
*TRADE MARK

Julia Miller does best on Rautensin—for no two hypertensives are alike.

When your geriatric, dyspeptic, underweight, or gallbladder patient doesn't respond to diet, the cause is frequently an inability to *utilize food*.

CONVERTIN furnishes the dietary catalysts necessary for efficient absorption in these individuals.

The specially layered construction of CONVERTIN provides selective release of ingredients to assure efficient absorption in the stomach and small intestine.

Each CONVERTIN Tablet provides:
a sugar-coated outer layer of:
Betaine Hydrochloride.....130.0 mg.
(Provides 5 minims Diluted Hydrochloric Acid U.S.P.)
Oleoresin Ginger.....1/600 gr.
Surrounding an enteric-coated core of:
Pancreatin.....62.5 mg.
(Equiv. 250 mg. U.S.P.)
Desoxycholic Acid.....50.0 mg.
DOSAGE: One or two tablets with or just after meals.
SUPPLIED: In bottles of 84 and 500 tablets.

B. F. ASCHER & COMPANY, INC.
Ethical Medicinals
KANSAS CITY, MISSOURI

cation may offer considerable difficulty in individual cases, because of scanty and contradictory etiological data or the fact that more than one etiological factor may be responsible for the defect, it does bring about a better understanding of the problem in relation to preventive measures.

Etiologically, mental defect can be divided into two large groups—endogenous or primary, and exogenous or secondary. In the exogenous group the defect comes chiefly from environmental factors. This group can be subdivided into types according to the causative agent—infectious, traumatic, toxic, and endocrine. On the other hand, an endogenous defect is determined mainly by those hereditary factors known as genes. The group includes conditions due to the combined action of many genes each of which alone would have an insignificant effect, or to the action of a single dominant or recessive gene.

HEREDITARY DEFECTS

Multiple genes.—Mental defects determined by multiple genes are "undifferentiated" in that they carry no specific physical distinction and are "aclinical" in that they show no clinical manifestations other than intellectual impairment. This group has also been designated by other terms: "residual" because it is composed of individuals who are left after a classification of specific forms; "subcultural" because so many of its members originate from low cultural environments; "familial" because of the high frequency of the condition in the patients' families. Since these cases can be diagnosed only by psychological and social adjustment criteria, differentiation between high-grade morons and dull-normal individuals may be difficult. While antisocial behavior and psychopathic traits occur in the group, they are far from universal.

Estimates of the incidence of undifferentiated mental defects run between 30 and 75 percent of all the mentally retarded, the lower figure probably running nearer to the facts. It includes defects of all grades, but high-grade morons predominate.

While the etiological factors determining the large number of undifferentiated cases of mental deficiency are still in dispute, it seems likely that they are similar to the factors responsible for general intelligence—in other words, genetic constitution. It seems reasonable to assume that most of these undifferentiated cases represent merely the lower part of the normal frequency-distribution curve of intelligence, known to statisticians as the Gaussian form. This means that a certain number of individuals are bound to appear in the range below the line indicating IQ 70. They are an integral part of the population as a whole, just as are individuals with superior intelligence with an IQ above 130. According to the curve, the majority of undifferentiated defectives are in the moron classification with IQ's between 50 and 70, and only a very few at the idiot level, with IQ's below 20—a picture which corresponds to observed fact.

Genetic constitution, however, is not the only source of all undifferentiated defectiveness, for environmental factors, such as subcultural milieu and poor hygienic conditions, undoubtedly play a causative role. The task of tracing the source of the defectiveness in individual cases is not easy, particularly when malnutrition and deprivation have been in the picture.

Single genes.—Some differentiated defects are determined by the presence of a single dominant gene transmitted from parent to child. Such defects are always traceable in the family history unless of a type that prevents reproduction. Frequently they turn up in severe form in alternate generations occurring in the intermediate generation only in incomplete form. Sporadic occurrences in families with no history of the defect are probably caused by a new mutation in a parental germ cell.

Data collected at Letchworth Village indicates that dominant genes probably account for only about 1 or 2 percent of all mental defects. These are always characterized by some physiological changes which make them classifiable into specific or clinically recognizable diseases. Among them are tuberosclerosis, neurofibromatosis, and nevoid idiocy—diseases in which mental deficiency is accompanied by skin lesions—and several forms of mental defect characterized by changes of bone structures.

There are also clinically recognizable defects caused by the presence of two

similar genes, known as recessive genes, one from each parent. Since persons of blood relationship are more likely to carry similar genes, such defects occur more frequently among the offspring of consanguineous marriages than in the general population.

In the great majority of the recessive cases the parents themselves are normal, being merely carriers of the gene, or, in genetic terms, heterozygous for the gene. The defect is characteristically distributed among 25 percent of the sibs, and is sharply distinguishable. While such defects are on the whole rare, they include a number of specific diseases: amaurotic family idiocy, a progressive and fatal disease accompanied by blindness which, according to type, may show up in infancy, childhood, or adolescence; gargoylism, a disease characterized by mental deficiency and grotesque bone changes; phenylpyruvic idiocy, the result of an inborn error in metabolism of an amino acid; hepatolenticular degeneration, a progressive form of mental deterioration caused by degeneration of nuclei at the base of the brain; and some forms of diffuse sclerosis, also a progressive disorder causing brain damage.

ENVIRONMENT-PRODUCED DEFECTS

A large but not yet clearly determined proportion of defectiveness comes from factors outside the hereditary constitution including infections, trauma, poison, glandular disorders, and physical or emotional deprivation. Rough estimates, based on unpublished data from a number of institutions indicate that such factors may account for at least half of the mentally retarded population in the country.

Infection. — Brain damage resulting from infection from the nervous system may occur in the womb or during infancy or childhood. The type of infectious agent, the severity of its attack, and the age of the child when attacked determine the degree of damage.

One of the most prevalent of such infections used to be syphilis, transmitted during gestation from an infected mother through the placenta to the fetus and resulting in brain damage to the fetus and later mental defect in the child. While syphilis still is responsible for a small percentage of all defectiveness, the proportion of infected children has already been reduced by venereal-disease control programs and undoubtedly will be further reduced in the future. Especially effective has been the increasing adoption of routine serological tests of pregnant women, prescribed by law in many States.

One form of severe mental deficiency comes from rubella infection (German measles) in the mother during the first 3 months of pregnancy. Besides the intellectual impairment resulting from fetal brain damage the rubella virus's attack on the fetus often produces congenital deafness, anomalies of the heart and eyes, and microcephaly (undersized head and brain).

Facts about the effects of other virus infections of the mother on the fetus are not so definitely established. It is possible that some other viruses may act in a manner similar to that of the rubella virus.

Brain fever is estimated to be responsible for the mental defects of 10 to 20 percent of all institutionalized defectives, according to Letchworth Village data. Caused by one of the encephalitis viruses or by a bacteria, such as the meningococcus of meningitis, it often strikes in infancy and childhood. While many children recover from it completely and others die, some recover with permanent impairments, the most common of which is mental defect. Measles, scarlet fever, chickenpox, whooping cough, influenza, and other communicable diseases common in childhood also occasionally leave brain damage.

Patients whose mental defectiveness has resulted from acute attacks of these diseases are usually referred to as postencephalitics. The degree of mental defect among them varies considerably with the individuals. Many of them exhibit a peculiar behavior pattern marked by episodes of overactivity, restlessness, impulsiveness, assaultiveness, and wanton destruction.

Trauma.—While accidents resulting in injury to the brain may sometimes occur in infancy or early childhood they are insignificant in comparison to injuries at birth or in the neonatal period as a cause of mental defect. Cerebral trauma during birth has been variously estimated to

for rapid clearing of the urine
Furadantin®
brand of nitrofurantoin, Eaton
In 30 minutes: antibacterial concentrations in the urine.
In 24 hours: a turbid urine is frequently clear.
In 3 to 5 days: complete clearing of the pus cells from the urine.
In 7 days: sterilization of the urine in the majority of cases.
With Furadantin there is no proctitis . . . no pruritus ani . . . no crystalluria . . . no moniliasis . . . no staphylococcic enteritis.
Tablets: 50 and 100 mg.
Furadantin Oral Suspension (5 mg. per cc.): Bottle of 4 fl. oz. (118 cc.)
Eaton
EATON LABORATORIES
NORWICH • NEW YORK
THE NITROFURANS— A UNIQUE CLASS OF ANTIMICROBIALS
PRODUCTS OF EATON RESEARCH

always in season

Sprains and strains . . .

Soreness and stiffness of muscles . . .

Neuralgia, arthralgia and kindred rheumatic pains . . .

Irritations and eruptions of the skin . . .

IODEX

cum Methyl Salicylate

combines the stimulating and metabolic effects of iodine in Iodex and the analgesic action of methyl salicylate.

Absorption through the skin may be aided by massage, heat or iontophoresis.

Samples and literature cheerfully sent upon request.

MENLEY & JAMES, LTD.

70 WEST 40TH ST., N. Y. 18, N. Y.

cause from 10 to 50 percent of all defectiveness. However, the incidence in institutionalized defectives does not seem to be above 20 percent. According to data gathered by the United Cerebral Palsy from one-half to two-thirds of the children in the general population showing evidence of birth injury are not mentally defective.

Difficult labor and prematurity are the most frequent causes of brain damage during birth, the former because of the risk of mechanical injury and the latter because of the immaturity of the brain. An immature brain is more prone to damage.

Brain damage at birth comes either by asphyxia or by hemorrhage. Asphyxia, which must be present for a relatively long period to produce irreversible damage, may result from premature separation of the placenta, cord complication, overdosage of the mother with analgesic drugs, or delayed breathing by the newborn. Hemorrhage, which may be within the brain or its envelopes, comes from direct injury during delivery—by forceps, or by a tearing of the tentorium, one of the membranes of the brain, in compression of the head during its passage through the pelvic canal.

Toxic causes.—Little is known about the effects of toxic factors transmitted from mother to fetus during pregnancy, but evidence exists for suspicion that there are several ways in which fetal poisoning, resulting in malformation and mental defectiveness, may occur. Eclampsia, a severe intoxication of obscure origin suffered by some pregnant and delivering women, may affect the child detrimentally. Some toxic drugs taken by a pregnant woman may also damage the fetus but what these are and how great the dosage must be to be damaging are still mysteries.

X-rays, on the other hand, are definitely known to be damaging to the developing central nervous system. Several cases are on record of mothers who after receiving deep X-ray therapy to the abdominal region during pregnancy have produced microcephalic children or children with other congenital abnormalities, including mental defect. However, improved knowledge of the effects of X-rays has resulted in the routine testing of women of child-bearing age for pregnancy before radiation, and thus in the reduction of defects from this cause.

Blood incompatibility between mother and child also has a toxic effect upon the child. This comes about most frequently as a result of the Rh factor, an entity present in the blood of about 85 percent of the population, but absent in the other 15 percent. When an Rh negative mother (whose blood possesses no Rh factor) carries an Rh positive baby, toxic substances develop which may cause damage to the fetal blood, liver, and brain. However, this condition is responsible for less than 1 percent of low-grade spastic defectives, as fortunately only 5 percent of Rh-positive children of Rh-negative mothers develop the disease, while some who do develop it recover completely.

Mongolism, or mongoloid idiocy, a condition with a characteristic physical appearance, may also be toxic in origin, although little is definitely known about its etiology. Some authorities believe that the condition appears in the fetus before the third month of pregnancy as a consequence of a variety of toxic conditions inherent in the mother and associated with advanced age, endocrine disorders, or pathological lesions of the uterus. Mongoloids comprise about 5 to 10 percent of all defectives. Their IQ usually runs between 15 and 40. Because these children are prone to infection, they have a higher mortality rate than other defective children.

Endocrine disorders.—While a certain percentage of mental defectives suffer from some glandular dysfunction, the proportion of defectiveness caused only by endocrine disorders is small. Cretinism is a form of mental defect definitely traceable to hypothyroidism or impaired function of the thyroid gland, either because of its lack of development or early destruction. This disease, which is also distinguishable by physical appearance, is endemic in areas where goiter is also prevalent, but it also occurs sporadically elsewhere. Dysfunction of the pituitary gland also causes mental defect, the most common type, Frölich's syndrome, being characterized by obesity, underdeveloped genitalia, and mild intellectual impairment.

Deprivation. — Emotional deprivation, frustrations, and insecurity may not only bring about a condition among normal

children resembling mental defect but may cause incorrect estimate of the intellectual abilities of high-grade defectives, especially those also physically handicapped. Pseudo-feeblemindedness is produced in normal children so deprived by an emotional blocking which responds to phychiatric treatment.

The most severe form of pseudo-feeblemindedness, infantile autism, is dramatic, if rare, evidence of the importance of emotional factors in the development of intelligence. Children so affected behave like idiots, do not talk, respond to stimuli, nor engage in any activity requiring intelligence, even though their intellectual capacity may be normal or better than average. Psychiatric examination shows that their apparent defect is a form of withdrawal.

The classical case of Kaspar Hauser exemplified the degree to which deprivation of the means of learning could impair intellectual development. Such extreme cases are not likely to occur today. Nevertheless, deprivation of cultural stimulation in some isolated communities still plays a role in producing the apparent low level of intelligence among the populace. More tragic are the effects of such deprivation on patients with disabilities interfering with academic learning. False diagnoses of feeblemindedness too often occur among children whose only impairments are in hearing, reading ability, word comprehension, minor motor handicaps, or other disabilities. In these children emotional factors are undoubtedly also contributing to the picture of apparent intellectual defect.

THE INDIVIDUAL

In spite of the growing knowledge of the causes of mental defects few specifics are available for their treatment or prevention. As the foregoing shows, mental retardation is not an entity itself, but a characteristic of a variety of conditions, each with a different cause. Moreover, in each form there is a wide range of intellectual ability.

Prevention for some forms may lie only within the scope of eugenic measures, though more scientific knowledge in the field of human genetics would be required before such could be confidently prescribed.

Greater possibilities for preventing the exogenous forms through medical and public-health measures may be expected to be realized as knowledge of intrauterine life and development increases.

While treatment in the strict medical sense can be applied only to a small number of mentally defective individuals, in the broader sense of care and training it can be applied to all. But such a wide variation of conditions exist among children with mental defects that what kind of care and treatment each receives must be determined individually in line with a prognosis based on an accurate diagnosis of the case. While the goal can rarely be cure, it can almost always be improvement or the achievement of the maximum intellectual and social functioning of which the individual is capable.

Functional Disorders of the Cardiovascular System

Bellergal®

Stabilizer of the entire Autonomic Nervous System

Ergotamine tartrate	*0.3 mg.*
Bellafoline	*0.1 mg.*
Phenobarbital	*20.0 mg.*

Adult Dosage: 4 to 6 tablets daily.

SANDOZ Sandoz
PHARMACEUTICALS
HANOVER, N. J.

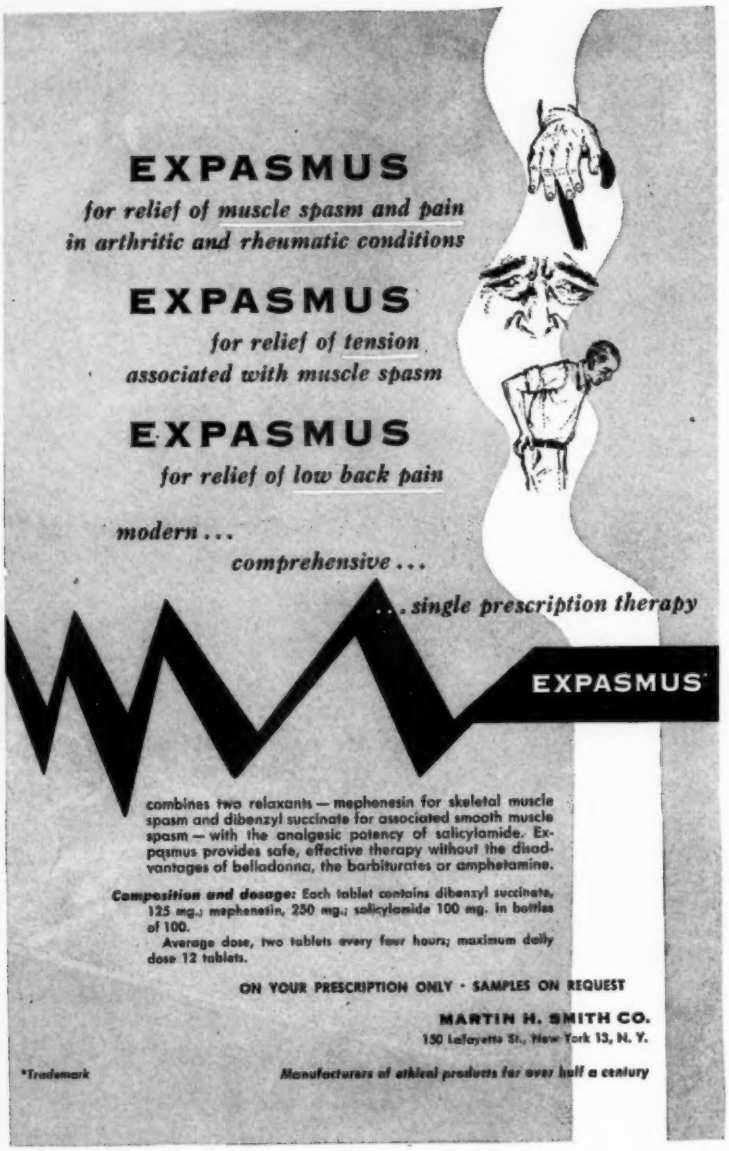
FATAL ACCIDENTS AMONG MEN AT THE WORKING AGES*

Accidents take more than 40,000 lives each year among men at the main working ages of life, that is, from 15 through 64 years. Only heart disease and cancer account for a greater number of deaths in this broad age group; in the 25 years of life from 15 through 39, more men die from accidental injuries than from any other cause.

Motor vehicle mishaps are the outstanding accident hazard for men under age 65, being responsible for nearly half of all the fatal injuries among them. Most of the men who lost their lives in motor vehicle accidents were occupants of the vehicle either as drivers or as passengers at the time they were injured. At the ages under 45, noncollision accidents—such as those in which vehicles overturn or run off the roadway—killed more men than did the collision of motor vehicles. At ages 45-64, however, accidents involving collisions took the greater toll.

Falls, ranking second as a cause of fatal injuries among insured men at the main working ages, accounted for one ninth of their total accident mortality. The death rate from this cause rises rather sharply with advance in age. As a result, the ratio of falls to all fatal mishaps in this experience increased from 3 percent at ages 15-24 to 21 percent at

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, September, 1954.


EXPASMUS
for relief of muscle spasm and pain in arthritic and rheumatic conditions
EXPASMUS
for relief of tension associated with muscle spasm
EXPASMUS
for relief of low back pain
modern...
comprehensive...
...single prescription therapy
EXPASMUS*
combines two relaxants — mephenesin for skeletal muscle spasm and dibenzyl succinate for associated smooth muscle spasm — with the analgesic potency of salicylamide. Expasmus provides safe, effective therapy without the disadvantages of belladonna, the barbiturates or amphetamine.
Composition and dosage: Each tablet contains dibenzyl succinate, 125 mg.; mephenesin, 250 mg.; salicylamide 100 mg. In bottles of 100.
Average dose, two tablets every four hours; maximum daily dose 12 tablets.
ON YOUR PRESCRIPTION ONLY · SAMPLES ON REQUEST
MARTIN H. SMITH CO.
150 Lafayette St., New York 13, N. Y.
*Trademark
Manufacturers of ethical products for over half a century

ages 45-64. Most of the fatal falls among men at the younger adult ages were due to falls from windows, ladders, scaffolds, roofs, and other high places. Past midlife, a considerable part of the mortality results also from falls on stairs and falls on the same level (such as those on floors or streets).

The loss of life from drowning, much of it sustained in sports and recreation, was almost as large as that from falls. Altogether, the three leading causes of accidental death—motor vehicles, falls, and drownings — were responsible for about seven out of every 10 deaths in this experience. A considerable part of the remaining mortality was due to burns and conflagrations, firearms, and poisonings by gases, solids, or liquids.

Viewing this insurance experience from another angle, we find that nearly one fifth of all the accidental deaths among men at the main working ages of life arose out of and in the course of employment; past early adult life the proportion was not far from one quarter. Injuries sustained in and about the home were responsible for an additional 10 percent of the accidental death toll among men at ages 15-64. The mortality from home accidents rose progressively with advance in age, reflecting in part the greater amount of time that older men spend around their homes.

It is encouraging that in the last 20 years material progress has been made in reducing the mortality from accidents among men at the main working ages of life. The reduction amounted to about one third between 1931-1933 and 1951-1953, the downward trend being more rapid in the first decade of the period than in the second. During these two decades the relative improvement in accidental mortality was greater at the later working ages than it was at the earlier ones. As a result, the disparity in the accident death rate between the youngest and the oldest age groups has narrowed considerably.

Despite the progress made, accidents still take an unnecessarily heavy toll of life among men at the main working ages. The White House Conference on Highway Safety and the President's Conference on Occupational Safety have given impetus to life-saving activities in these fields. But the attitudes and behavior of individuals remain the focal point of the safety problem. Many lives of both men and women would be saved each year if people obeyed the traffic laws strictly, exercised caution in recreational activities, observed the rules of safety on their jobs, and kept their homes as free from accident hazards as possible.

SOME THOUGHTS ON CANCER RESEARCH*

By R. Keith Cannan, D.Sc.†

The objective of cancer research is the control of cancer in man. This statement would appear to be so obvious as to require no elaboration. The inquiring layman, however, who surveys contemporary programs of research sponsored by agencies interested in the control of cancer may well find occasion to pause. He will observe that a large proportion of the work currently in progress is being conducted in laboratories rather than at the bedside, and he will note also that much of it is concerned with problems that seem to bear no apparent relation to human cancer. He may well wonder whether investigators are losing sight of the medical woods in their zest for climbing their own particular scientific trees.

Why is cancer research diffused so widely over the face of medical science, and why is much of it so remotely related to the cancer patient? The question is a pertinent one and merits thoughtful answer.

In this matter, as in most critical appraisals, it is well to begin by laying down some basic propositions. Two will suffice for the argument that follows.

The first proposition is that cancer is a disease of individual cells. The weight of evidence forces the conclusion that a tumor is merely the visible sign of the presence in a tissue of colonies of aber-

*Reprinted from *Public Health Reports*, November, 1954.

†Dr. Cannan is chairman of the Division of Medical Sciences, National Research Council. His discussion of cancer research was prepared at the suggestion of the National Cancer Institute, Public Health Service, and is based on his testimony at the health inquiry hearing of the Committee on Interstate and Foreign Commerce, House of Representatives, October 2, 1953.

rant cells that have broken through the normal restraints on cell growth and are multiplying without regard to the health of the tissue from which they came or the economy of the organism as a whole. They are, however, not merely normal cells "on the loose." They are specifically and irreversibly modified cells. They are disordered rather than disorderly. This conclusion is implicit in the fact that their abnormal characteristics are such an intimate part of the cells that they are passed on to their progeny in the course of cell division. Cancer is a self-multiplying disorder of cells and therein lies its malignancy.

The second proposition is that the living cell is a chemical machine. A cell is recognized microscopically by its characteristic structure, but it is much more than an inert structure. It is a process. A living cell is a "becoming" rather than a "being." It lives by reason of a constant flux of chemical change whereby energy is mobilized for growth and multiplication and for the contribution of the individual cell to the activities of the organism as a whole. The structure of a cell, like that of an inanimate machine, is simply an organization of parts adapted to the purpose of converting energy into specific processes in an orderly and efficient manner. Each type of cell has its own peculiar structure designed to effect its own particular processes. Each type of cell has its own pattern of chemical activity. Change in structure or change in physiological function is a reflection of some basic change in this complex of chemical reactions. In the last analysis the understanding and control of cellular activity is a biochemical problem.

A BIOCHEMICAL PROBLEM

If these two propositions are accepted, it follows that a malignant cell must differ chemically from a normal cell because it behaves differently. Since it continues to multiply under conditions in which its neighbors have ceased to do so, the cancer cell must be pursuing a pattern of chemical behavior different from that of the community of normal cells. If this is so, then the chief hope for the control of malignant growths that are beyond the reach of the surgeon's knife or the X-ray beam is the control of their chemical reactions.

Ten or fifteen years ago this conclusion would have been an affirmation of faith without proof because so little was known of the intimate chain of chemical events within the cell. Today, it may be asserted with some confidence that there is accumulating evidence that malignant cells do differ chemically from normal cells. This information has not yet provided us with a workable formula for controlling the growth of cancers, but it does justify the hope that chemical means to do so will be found.

The emphasis which has been placed on the biochemical approach to cancer does not detract in any degree from the importance of biological studies that do not involve chemical techniques. The relation of hereditary factors to cancer, the transplantability of cancers, the transmission of tumors by viruslike agents, the growth of cancer cells in tissue culture, the influence of hormones on susceptibility to the growth of tumors, the attempts to develop antibodies to malignant cells—all these and many other types of study are essential contributions. Without them the biochemist would have nothing to investigate. In the final issue, however, cancer is a biochemical problem, and the ultimate objective of those who are pursuing the biological avenues of approach indicated above must be to find chemical descriptions for the phenomena that they observe.

OBLIGATIONS OF MEDICINE

Medicine recognizes two major obligations. The first is the diagnosis and cure of disease as and where it is encountered. A more far-reaching obligation, however, is the prevention of disease by the disclosure and control of its causes. Much of the contemporary research in the field of cancer appears to be directed toward the search for the causes of malignant growth rather than toward the refinement of the therapeutic measures that offer the greatest immediate promise. There has been some criticism of this distribution of effort, but the criticism ignores the fact that the two approaches are not competitive but complementary. The worker in the laboratory is constantly developing new ideas, devising new tools, and exploring new concepts, which he refines and passes on to the clinical investigator. The clinical investigator, as constantly, is testing these ideas and applying these tools in the clinic and is passing

. . . no two hypertensives are alike

Tim Jones must stay on Rauvera
Diagnosis: Moderately severe, chronic, fixed hypertension; arteriosclerosis.

T. J., nervous tense executive, used to be on Veratrum products, but the high dosage necessary to control his symptoms caused annoying nausea and occasional vomiting. Now he is doing well on Rauvera (1 tablet t. i. d. after meals). The ***combination therapy*** of 1 mg. alseroxylon and 3 mg. alkavervir per tablet of Rauvera is the best medication for him, because it reduces his need for Veratrum and successfully manages his Grade III type of hypertension.

On Rauvera his blood pressure dropped promptly, his headaches disappeared, his pulse rate slowed, he is in good spirits. No toxic effects were noted.

Rauvera® is a DORSEY preparation. Supplied in bottles of 100, 500, and 1,000 tablets.

Smith-Dorsey • Lincoln, Nebraska • A Division of The Wander Company

Tim Jones must stay on Rauvera—for no two hypertensives are alike.

improve
capillary
resistance
in prevention
and treatment of
capillary fragility
capillary hemorrhage
vascular accidents

C.V.P.

(CITRUS FLAVONOID COMPOUND WITH VITAMIN C)

may protect against abnormal bleeding and vascular accidents in...

- hypertension
- retinal hemorrhage
- diabetes
- radiation injury
- purpura
- tuberculous bleeding

Five years of laboratory and clinical investigations establish the complete safety and value of C.V.P. in increasing capillary resistance and reducing abnormal bleeding due to capillary fragility.

C.V.P. provides natural bio-flavonoids (whole natural vitamin P complex) derived from citrus sources—potentiated by vitamin C—which act synergistically to thicken the intercellular ground substance (cement) of capillary walls, decrease permeability... and thus increase capillary resistance.

"Many instances of hemorrhage and thrombosis in the heart and brain may be avoided if adequate amounts of vitamin P and C are provided."

each C.V.P. capsule provides:

Citrus Flavonoid Compound*	100 mg.
Ascorbic Acid (C)	100 mg.

*(water soluble whole natural vitamin "P" complex, more active than insoluble rutin or hesperidin)

bottles of 100, 500 and 1000 capsules

Professional samples and literature on request.

USV u. s. vitamin corporation
(Arlington-Funk Laboratories, division)
250 East 43rd Street, New York 17, N.Y.

NEW! C.V.P. SYRUP, pleasant tasting, especially useful to help prevent bleeding after tonsillectomy. Each 5 cc. (one teaspoonful) equivalent to one C.V.P. capsule.

back new questions, thereby channeling the work in the laboratory. A remarkable aspect of clinical investigation today is the speed with which new knowledge, garnered in the laboratory, receives clinical appraisal. In point of fact the seekers after causes and the seekers for diagnostic tests and therapeutic measures may pose their questions differently, but they find themselves exploring the same ground—the ground of the biochemistry of the cell. It is quite probable that the next major advance in chemotherapy may arise from some observation made in the course of an academic study of the cause of a particular animal tumor. It is also possible that the next new idea about the causes of malignant growth may emerge from clinical studies at the bedside or in the field.

KNOWN CAUSES

A few extraneous causes of human cancer are known. They include several types of radiation and a heterogenous group of chemicals comprising the salts of a few metals, certain curious hydrocarbons, some tars, and a few intermediates in the manufacture of dyes. This is a motley assembly. These agents are known to interfere in one way or another with the chemistry of cells, but their chemical effects exhibit no common characteristic which would explain their unique property of promoting cancerous growth.

To what extent human cancer is caused by exposure to environmental cancerogens is uncertain. Those enumerated above are relatively minor causes. On the other hand, the alarming increase in lung cancer in males during the past three decades has alerted medicine to the fact that civilized man is increasingly surrounding himself with an unnatural chemical environment whose cancerogenic potentialities have not been explored. Intensive studies of the possible relation of the incidence of particular types of cancer to exposure to chemical contaminants by contact, inhalation, or ingestion are badly needed.

SUSCEPTIBILITY

Although the known environmental cancerogens are of clinical importance only with respect to limited groups of people who may be subject to undue exposure to one or another of these agents, they have proved to be immensely valuable tools in the laboratory because they make possible controlled studies of the early and late stages in the progress of malignant growth. Such studies have established the fact that the development of a tumor depends not only on the presence of an inciter but also on the susceptibility of the exposed cells. There are wide differences in sensitivity to a particular cancerogen of the various types of cell in a single animal and also of a single type of cell in different species and, even, in different strains of the same species. To add to the confusion the relative susceptibilities of particular cell types to a series of cancerogens are quite variable. Such observations, coupled with the knowledge of the wide differences in the natural histories of the many types of human tumors lead to the inescapable conclusion that cancer is not one but many diseases and can have many causes.

It must be acknowledged that the term "susceptibility" is a cloak for ignorance. In part it represents the inherent resistance of the cell to the action of the inciter and, in part, the extent to which the body as a whole is able to restrain the tendency toward disordered growth that is presumably invoked by the inciter in susceptible cells. The hormones, and possibly other regulatory mechanisms of the body, are involved in the execution of these restraints. Whatever may be the complex of processes that determine the susceptibility of a cell to a particular inciter, the processes must be chemical in nature because the cell is a chemical machine.

Insofar as human cancer cannot be shown to be environmental in origin, the investigator must look within the body for its causes. In this difficult quest there are few leads. One can only surmise that there must arise in the body of the host some cancerogenic agent which evokes malignant changes in susceptible cells. This inciter may be a product of some chronic disorder of metabolism, some spontaneous distortion of the chemical mechanisms of heredity, or, possibly, an ill-defined agent comparable to the viruslike bodies that are known to be associated with certain animal tumors.

The argument that has been developed above reduces to the simple statement that the goal of the seeker after causes is the disclosure of chemical anomalies antecedent to the overt development of

malignant cells. As and when these are uncovered, it will be possible to develop rational approaches to their therapeutic correction or control. In the meantime, attempts to improve present methods of diagnosis and treatment cannot be neglected. The best must not be allowed to be the enemy of the good.

PRESENT HOPE

Cancer can be cured if it is localized and accessible to the surgeon or the radiologist. The urgent challenge of cancer is the detection and control of hidden and diffuse malignancies. Present hope lies in chemotherapy. Chemical agents must be sought which will be carried in the blood stream to the cancer cells and will destroy them or inhibit their growth without disrupting the essential functions of normal tissues.

Faced with millions of available compounds, the investigator will not attempt to proceed on any hit-or-miss basis. He seeks out differences in the chemistry of normal and malignant cells in the hope of identifying cellular reactions which are more sensitive to chemical interference in cancer cells than in normal cells. At present the direction taken in most chemotherapeutic studies stems from the argument that, because malignant cells multiply rapidly, the synthetic reactions associated with cell division are the most promising targets. On this basis it is possible to make a rational approach by selecting as potential interfering compounds a restricted group of chemicals that may be expected to interact with the cell constituents whose synthesis seems to control the division of the cell.

Even so the problem is proving to be highly complex. Each type of tumor in animals shows its own range of sensitivities to a series of drugs. Moreover, the same type of cancer in different species varies in its sensitivity to a particular chemotherapeutic agent. Evidently, each type of tumor in man must be investigated as a separate therapeutic problem. It is unlikely that a single miracle drug will be found that will control all of the major cancers that afflict mankind. On the other hand it becomes increasingly probable that medicine will develop a battery of drugs each of which will have therapeutic value under limited conditions.

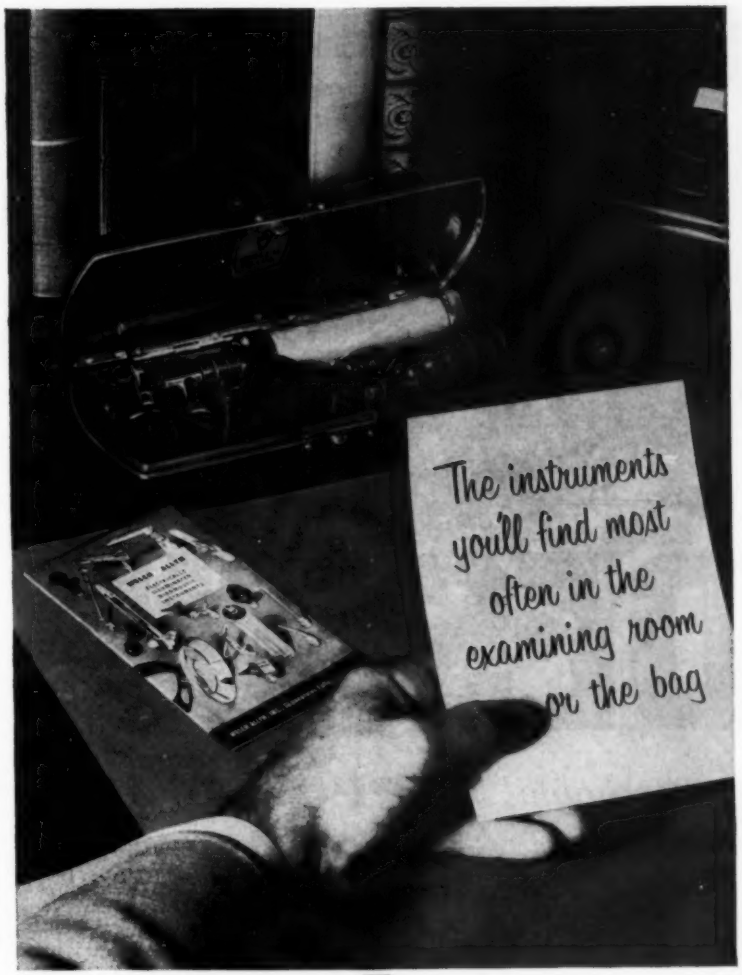
In the control of human cancer much emphasis has been placed on the value of early diagnosis. Here, again, it is the buried and diffuse tumor that is the urgent problem. The hope is that the chemical disturbances that are presumed to be associated with the precancerous or early cancerous states are reflected in chemical changes in the body fluids and that means of identifying the latter can be devised. As in the case of chemotherapeutic agents it is unlikely that a single test will betray all types of cancer, but there is reason to hope that a series of tests of limited specificity will be evolved.

CONCLUSIONS

In conclusion, one may properly ask the question why the intensive study of the chemistry of cancer has been so long delayed. The answer can only be that the solution of a scientific problem requires tools appropriate for the task. With respect to the biochemistry of cancer, progress waited upon the development of instruments and techniques sensitive and gentle enough to probe the chemical activities of small groups of cells while they are alive and functional. These are now at hand. Specifically, the emergence of radioactive isotopes as molecular tracers has been the master key that is unlocking the doors of the biochemistry of the cell.

Notable advances have been made during the past decade in all avenues of cancer research. However, the most promising aspect of the contemporary scene is not the accumulation of isolated pieces of information but the fact that the many avenues to knowledge are rapidly drawing together and finding common ground in biochemistry. The biochemist and the biophysicist have now refined their techniques to the point that they are ready to join hands with the clinician, the geneticist, the cytologist, the microbiologist, and the endocrinologist. When scientists using different vocabularies reach common ground and learn to employ the same language, a notable intellectual synthesis has been achieved.

It may be well to emphasize that the timing of this larger synthesis was not at the command of investigators within the field of cancer studies. Its emergence depended upon developments in all the sciences ancillary to medicine. The future of cancer research does not lie in the hands of any group of the elect. It is determined by the progress of science as a whole. Herein lies a moral for those



WELCH ALLYN

Skaneateles Falls, N. Y.

DESIGNERS AND MANUFACTURERS OF DIAGNOSTIC INSTRUMENTS FOR 39 YEARS

prescribe a full measure of comfort for anorectal patients with

DESITIN®

hemorrhoidal **SUPPOSITORIES**

with cod liver oil

In boxes of 12 foil-wrapped suppositories

DESITIN SUPPOSITORIES quickly soothe, protect, lubricate the distressed anorectal mucosa to provide.....

- **gratifying comfort in hemorrhoids** (non-surgical)
- **rapid, sustained relief of pain, itching and spasm without styptics, local anesthetics or narcotics, therefore do not mask serious rectal disease**
- **reduced engorgement, bleeding • safe, conservative**

samples yours for the asking

DESITIN CHEMICAL COMPANY • 70 Ship Street, Providence 2, R. I.

who administer the affairs of medical research. To attempt to encourage the study of a single disease as phenomena isolated from the main stream of medical and biological knowledge is the surest way to impede progress.

WHERE FATAL ACCIDENTS OCCUR*

One of the difficulties facing the safety movement is that accidents occur in a wide variety of places. Fatal accidents take place in and about the home, in industrial plants and factories, on streets and highways, in places of recreation—in short, wherever people live, work, play, or travel. One of the striking facts is that transport accidents account for approximately as many deaths as all nontransport accidents together.

Whatever the place of occurrence, accidents took a heavier toll of life among males than females. In industry—factories, workshops, mines, and quarries—and on farms, deaths from accidents were almost exclusively among males, reflecting the greater number of men employed in these places and the greater hazards attending their work. For transport accidents, the ratio of the male to female death rate was about 3½ to 1. Even for home accidents the excess male mortality amounted to about a third. For all accidents combined, the death rate for males was about three times that for females among these Industrial policyholders.

Injuries sustained in motor vehicle accidents accounted for 85 percent of the deaths from transport accidents among males and for an even larger proportion among females. The other transport deaths were due largely to water transportation, railroad, and aviation accidents. In addition, there was a relatively small number of deaths in mishaps involving bicycles, streetcars, and other road vehicles. It is of interest that an appreciable part of the fatal injuries in motor vehicle, water transport, and railroad accidents among men arose out of and in the course of employment.

In industry, falls and injuries involving machinery outranked every other type of fatal accident. Among males, falls constituted somewhat over one fifth of the deaths, and machinery accidents a somewhat smaller proportion. An appreciable number of fatal industrial injuries among males—13 percent—were due to burns other than in conflagrations. On farms, machinery was the major hazard; most of the deaths from this cause resulted from the overturning of tractors operated in agricultural work. The rather common use of firearms in rural areas undoubtedly accounted for the fact that such weapons ranked second in this experience as a cause of accidental deaths on farms.

Falls are by a considerable margin the leading cause of accidental death in and about the home. In this experience they accounted for two fifths of fatal home injuries among males and for virtually one half of such deaths among females. These proportions would unquestionably have been higher if policyholders at ages 75 and over had been included in this study, because fatal falls are largely concentrated at the older ages. Conflagrations and burns by other means were also leading causes of accidental death, together being responsible for more than one quarter of the fatal injuries in and about the home among males and for fully one third of those among females. Among males the toll from conflagrations exceeded that for other types of burns, but for females the situation was the other way around. Gas poisoning, too, took an appreciable number of lives in each sex.

Falls were particularly significant in fatal accidents on streets and highways (other than transport), in public buildings, and in resident institutions.

A large proportion of the fatal injuries classified as occurring in places used for sports and recreation, rivers, woods, and the like was sustained in swimming, hunting, and other sports activities. Drowning alone accounted for about two thirds of all the deaths in this residual category. Falls and firearm accidents were also fairly prominent in the total, particularly among males.

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, November, 1954.

HEALTH EDUCATION FOR OUTPATIENTS*

By Mary Lou Skinner, M.P.H., and Mayhew Derryberry, Ph.D.†

(Continued from January JOURNAL*)*

The Patient's Response

It saves time and makes for effective education to know the patient's opinion. After all, the patient acts on his own opinion whether it is correct or not. A simple well-planned interview with the patient usually will obtain the necessary information. When patient opinion is known, it is possible to plan an educational program which will hit the target more accurately.

Most patients realize that they are not competent to judge medical care, but they become impatient when their wishes and ideas are ignored. Their progress toward recovery and in education can be delayed by rules and procedures they do not understand and by indifferent personnel. Positive education is not apt to occur in a discontented person. Many people lose their dissatisfaction when they realize that the clinic is sufficiently interested in them to find out what their opinion is. One study of patient opinion found that most satisfied patients complained about not having the hospital schedule explained (*4*). Is this worth exploring in outpatient clinics?

The interviewers in this same poll were startled by one complaint which may have significance for outpatient clinics. Patients who were generally well satisfied did not feel that they had received enough information about how to care for themselves at home. The poll-takers concluded that "it was evidence of the failure of nurses and doctors to seize opportunities for health teaching; as well as showing a lack of interest in the patient as a person who will continue to exist and have health problems related to his illness even after he leaves the hospital. It could show, too, a lack of integrated planning with allied health agencies for followup care."

DIRECT EDUCATION PROGRAMS

The "diagnostic" process (*5*) of telling people things is not the most successful or satisfying way of working educationally with them—it is often dangerous, and its effect cannot be foreseen. It would seem preferable to adopt the "therapeutic" process of helping people to recognize things for themselves. However, since many shared attitudes are derived from the teacher-pupil experience of academic life, the attitude that one educates people by doing things to them is carried over into, and dominates, the educational relationships to patients. The difference in the diagnostic and therapeutic processes is between doing things "to" or "for" people and doing things "with" them. These ways of thinking affect the indirect and direct education that is done.

Printed literature, exhibits, and information racks are useful tools in an educational program, but the extent of their effectiveness depends on thoughtful planning for their use. The Veterans Administration Hospital in Rutland Heights, Mass., used to give packets of pamphlets on tuberculosis to every new tuberculosis patient admitted. They lay untouched on the bedstands. However, when racks were put in the wards where the men could make their own selections, pamphlets began to disappear, in considerable variety and number.

Another, and even more significant use of health literature, is that reported by Schwartz (*6*). The Cornell University Medical Center outpatient department in New York City started using pamphlet racks with a wide range of subject matter. Some of the staff were cynical.

Why child guidance leaflets in an adult clinic? they asked. Who on those benches requires prenatal care? A glance would show that the average age is 60. But patients waiting on outpatient benches, like people everywhere, have families. They did not discard family problems or interests at the clinic door, and the pamphlets disappeared.

*Reprinted from *Public Health Reports*, November, 1954.

†Mrs. Skinner is health education consultant in the Public Health Service, Department of Health, Education, and Welfare, Region II, New York City. Dr. Derryberry is chief, Health Education Services, Division of General Health Services, Public Health Service. Their joint paper was presented to the outpatient session of the Tri-State Hospital Assembly at Chicago, Ill., on March 4, 1954.

when joints need "oiling"

Pabirin ... safest of the antirheumatic salicylate-paba combinations

because ... Pabirin does not produce salicylism even with heavy daily requirements. *High* blood levels are maintained with *low* salicylate dosage. It contains well-tolerated acetylsalicylic acid, the most effective of the salicylates. Pabirin is sodium- *and* potassium-free. A therapeutic amount of 300 mg. of ascorbic acid in the average daily dose of six capsules offsets depletion of vitamin C by salicylates.

And effective because ... The synergistic effect of acetylsalicylic acid and PABA and the retarding action of PABA on salicylate excretion ensure high and sustained blood levels. Rapidly disintegrating capsules provide fast absorption and pain relief.

Pabirin is a DORSEY preparation.

Each capsule contains:
Acetylsalicylic acid ..... 5 gr.
Para-aminobenzoic acid ..... 5 gr.
Ascorbic acid ..... 50 mg.
Average dose: 2 to 3 capsules 3 or 4 times daily.
Supplied: In bottles of 100, 500 and 1,000 capsules.

Pabirin®

Smith-Dorsey • Lincoln, Nebraska • A Division of The Wander Company

The educator was still not satisfied, and another approach was tried. This time only one copy of each leaflet was made accessible.

It was clearly marked "Please do not remove this booklet from the clinic. If you think a copy will help you, talk with the nurse about obtaining one." These pamphlets were mixed with copies of the *Saturday Evening Post* and other magazines. To the surprise of the clinic staff, there were 136 requests for the leaflets in the first 4 weeks. Pamphlets in hand, the patients came and asked for information. The conferences which followed brought to light all manner of problems not directly related to the patient's reason for coming to the clinic. Transfer to physicians of information important to the treatment of the individual patients was a valuable result.

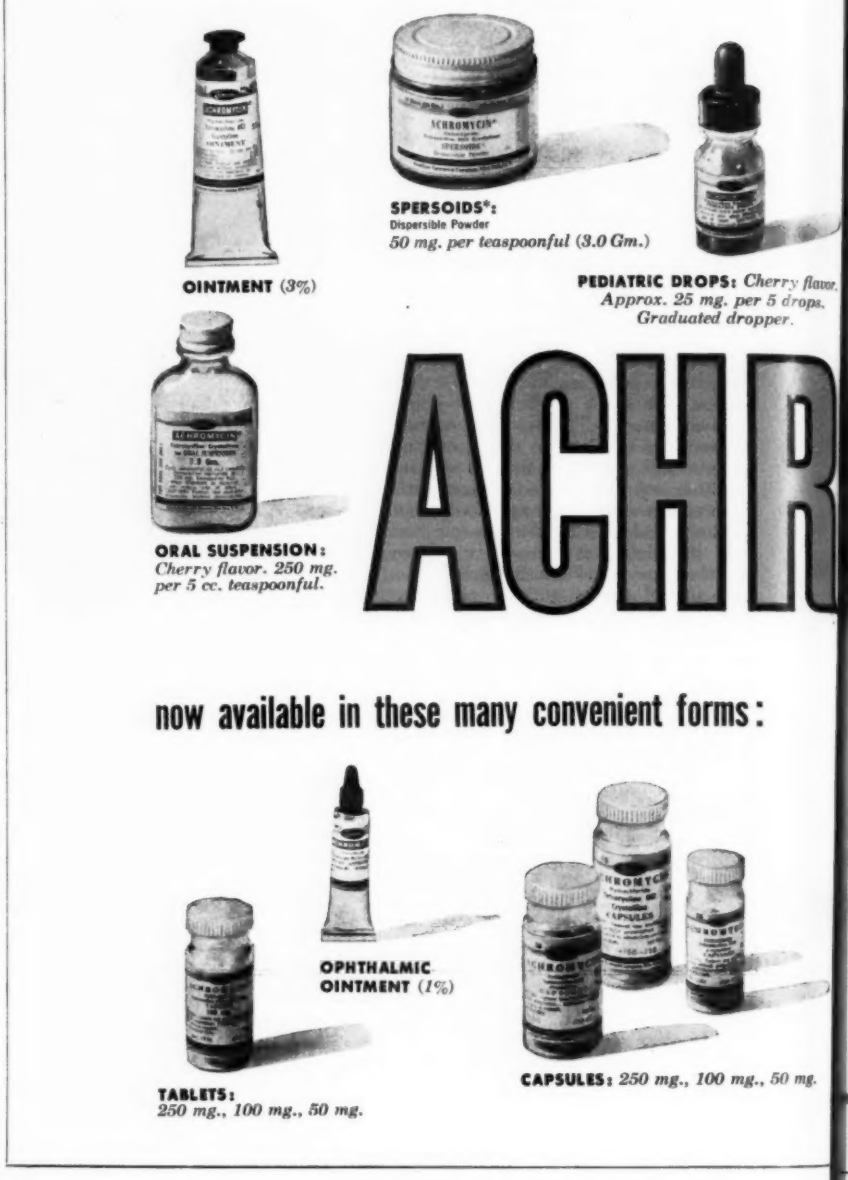
Advising and planning with patients and their families on what to expect in connection with medical therapy can produce favorable educational results. The study on hospitalization of children for tonsillectomies (*7*) conducted by the Albany (N. Y.) Medical College Department of Pediatrics is an example of such anticipatory planning. Among other things, this study showed that young children resented a jab with a hypodermic needle. The child was told that the only jab he had to take was the finger prick for a hemoglobin reading. The staff found that if young Philip knew in advance that his finger would receive a slight prick, such as he gets dozens of times playing, followed by the appearance of a round bead of his own beautiful red blood, he was likely to watch without anxiety while the bead of blood rose in a little glass tube, just as he had been told it would. Mother might stand by trying to keep from shuddering, but Philip was fascinated. This is health education of real significance.

Any number of people, given the opportunity, will welcome the chance to help with the clinic's education program. It is not at all necessary for the clinic to do it alone. If mimeographed maps or guides to the clinic routine are needed, volunteers can solve the problem. If the staff is too busy to take time to talk with foreign-born folks who struggle with English, the local adult education center may be able to supply volunteers from its foreign language classes.

If time will not permit inservice education of staff, a plan used in Boston may be the solution. There, 9 hospitals and 11 official and voluntary agencies set up a committee on outpatient education to plan ways for improving educational practices. Educators are members and suggest improvements for the staffs to try. Other services for which the hospital clinics do not have time to recruit independently are provided in other ways.

Volunteers provided the Lubbock Memorial Hospital in Lubbock, Tex., with pamphlet racks and kept them filled; they also provided transportation for the indigent. While doing this, they learned about another need and have helped the hospital develop a Negro prenatal clinic. Now, they are working on community understanding of welfare problems which affect the hospital. Thus, a double educational goal is being served. Not only are the patients in the hospital learning, but the volunteers have discovered many opportunities for effective action.

Group activities is one of the more significant methods that can be used by clinics to provide learning opportunities. This method is being tried in a wide variety of situations. Group discussions with parents in child health conferences at the Lillian Wald Health Station in New York City (*8*), group discussion with parents and families of premature babies and of children with rheumatic fever at Grace-New Haven Community Hospital, New Haven, Conn. (*9*), the group work with diabetic patients carried on in Boston by the city hospital and the Diabetes Field Research and Training Unit of the Public Health Service, and the group work with obese patients at Herrick Hospital in Berkeley, Calif., indicate some of the experiments. Group work with patients who have had coronary attacks has been proposed. Alcoholics Anonymous is working closely with some hospital clinics in group discussions with alcoholic patients. Group methods of education can be used to help parents of handicapped children learn how to work more successfully with their own children (*10*).


SPERSOIDS*:
Dispersible Powder
50 mg. per teaspoonful (3.0 Gm.)
OINTMENT (3%)
PEDIATRIC DROPS: Cherry flavor. Approx. 25 mg. per 5 drops. Graduated dropper.
ORAL SUSPENSION: Cherry flavor. 250 mg. per 5 cc. teaspoonful.
ACHRO
now available in these many convenient forms:
OPHTHALMIC OINTMENT (1%)
TABLETS: 250 mg., 100 mg., 50 mg.
CAPSULES: 250 mg., 100 mg., 50 mg.

INTRAVENOUS:
500 mg., 250 mg., 100 mg.

SOLUBLE TABLETS:
50 mg.

INTRAMUSCULAR: 100 mg.

*

ROMYCIN

Tetracycline Lederle

EAR SOLUTION (0.5%)

ACHROMYCIN, the new broad-spectrum antibiotic, is now available in a wide range of forms for oral, topical and parenteral use in children and adults. New forms are being prepared as rapidly as research permits.

ACHROMYCIN is definitely less irritating to the gastrointestinal tract. It is more rapidly diffusible in body tissues and fluids. It maintains effective potency for a full 24-hours in solution.

ACHROMYCIN has proved effective against beta hemolytic streptococcic infections, *E. coli*, meningococci, staphylococci, pneumococci and gonococci, acute bronchitis, bronchiolitis, pertussis and the atypical pneumonias, as well as virus-like and mixed infections.

* REG. U. S. PAT. OFF.

ERLE LABORATORIES DIVISION AMERICAN Cyanamid COMPANY Pearl River, N.Y. Lederle

"Group activities" in this context does not mean classes in the traditional lecture form; it means, instead, ways of bringing people together to create situations in which they feel free to discuss their own reactions about the problems in question. They learn from each other and give support to each other. The classic study (*11*) with mothers of young infants on feeding orange juice and cod liver oil, done at the University of Iowa Hospital, brought out clearly the advantage of decision-discussion methods. In such group discussions, incorrect ideas are likely to be rejected by the group; people are strengthened by group attitudes of their peers and supported in changing or in tentative new convictions.

Leadership for this kind of group discussion is a skill. Only recently has it been taught in school. Moreover, new studies in group discussion methods are going on all the time. The clinic staff which is interested in exploring this method would be well advised to consult with local resources for help in adapting these skills to their special needs.

ANOTHER CHALLENGE

Not many in public health or hospital administration are specialists in education methods or have the skills of social scientists. As in the medical field where general practitioners sometimes fail to recognize a symptom which a specialist would spot at once, so in the education field it is easy to miss significant opportunities for education. Social scientists can be called on for help on problems in these fields. More and more hospitals are asking educators or sociologists to observe their practices and to suggest more effective ways of working. The demand is growing for consultation and service from anthropologists, health educators, adult educators.

These different professional groups will not provide a common body of accumulated knowledge with ready-made answers. The professions are advancing together into new, experimental, and more profound ways of working with people. Although these newer methods are not so well formed or definitive as the older academic ways of teaching, they can, when wisely used, offer greater satisfaction. Imaginative people in a clinic, who take their responsibilities thoughtfully, will be challenged to find out from day to day what the patients are learning and to enhance that knowledge positively.

It is important for everyone to become more familiar with the nature of the cultures and the ways of life of others: their goals in life; their values, beliefs, traditions, customs, and taboos with respect to health and illness. More must be known about the objectives for which people are willing to strive and conversely, more must be understood about the aspects of life which mean very little to them. These understandings are not easy to acquire. Since understanding is limited or not always possible, perhaps respect for differences is an attitude to nurture.

The only people who are really going to do anything for the patient are the persons who work with him. Not everyone is equally ready to learn about health at all times. But there are times in a lifetime when an individual is ready. One such time is when he comes to the outpatient clinic for service.

REFERENCES

(*1*) Nyswander, D. B.: The implications of new educational concepts for the prevention of prematurity. California's Health 10:17 (July 15, 1952).

(*2*) Zborowski, M.: Cultural components in responses to pain. J. Social Issues 8:4:16-31 (1952).

(*3*) National Tuberculosis Association: Report of the Denver hospital study. New York, N. Y., The Association, 1952, 30 pp.

(*4*) Randall, M.: Polling patient opinion. University of Minnesota Hospital Staff Meeting Bull., Vol. 19, No. 9 (December 5, 1947).

(*5*) Wilson, A. T. M.: Implications of medical practice and social case-work for action research. J. Social Issues 3:11-28 (Spring 1947).

(*6*) Schwartz, D.: Health promotional literature for clinic patients; An experiment by the Nursing Service of the New York Hospital, Cornell Medical Center, Out-Patient Department. Am. J. Pub. Health 43:1318-1323 (1953).

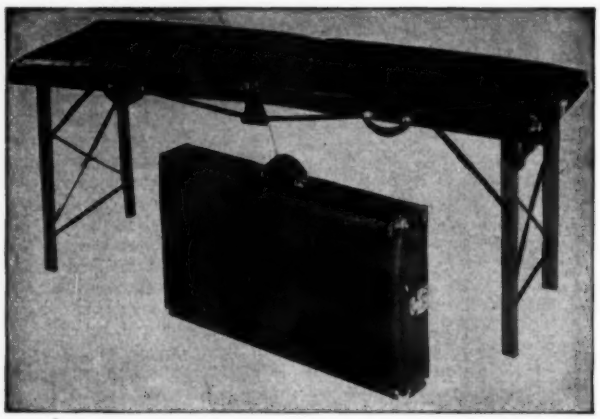
(*7*) Winkley, R.: When a child must go to the hospital. Child 17:34-36 (November 1952).

(*8*) Wishik, S. M.: Parents' group discussions in a child health conference. Am. J. Pub. Health 43:888-895 (1953).

(*9*) Kirchner, A.: Parents' classes in a maternity program. Am. J. Pub. Health 43:896-899 (1953).

(*10*) Milliken, S.: Group discussion of parents

IDEAL FOLDING TABLE



Well constructed, strong.
Will not tip or shake.
Easy to open and close.
Length 69". Width 22".
Height 27½". Weight 32 lbs.
Walnut finish.
Simulated leather covering.
Heavy standard padding.
(Shipping weight 35 to 37 lbs.)

FOR HOME **AND OFFICE**

Price $40.00

(Paratex and felt) 2" Paratex padding $10.00 additional

Unconditional guarantee on workmanship and materials. All items shipped f.o.b. from Factory in Kirksville, Mo. Cash must accompany orders.

American Osteopathic Association

212 E. Ohio St. Chicago 11, Illinois

of handicapped children from the health education standpoint. Am. J. Pub. Health 43:900-903 (1953).

(*11*) Radke, M., and Klisurich, D.: Experiments in changing food habits. J. Am. Dietet. A. 23:403-409 (1947).

INFECTIOUS HEPATITIS—A GROWING HEALTH PROBLEM*

The past few years have witnessed a sharp rise in the reported number of cases of infectious hepatitis, an acute virus infection usually accompanied by jaundice. Although the disease frequently escapes detection, somewhat over 50,000 cases† will probably be reported in the United States during 1954, or about one and one-half times the number reported the year before, and nearly three times the total for 1952. The major part of this increase undoubtedly reflects merely the better recognition and more complete reporting of cases. However, some of the rise in the incidence of the disease probably is real. On the basis of its reported incidence, infectious hepatitis now ranks among the leading communicable diseases. Fortunately, only a small proportion of the cases terminate fatally. In 1952, there were only 794 deaths from the disease reported in the United States, equivalent to a death rate of .5 per 100,000 population.

Although infectious hepatitis strikes at every period of life, its incidence is highest among school-age children. The accompanying table shows the age distribution of cases in several States for which such data are available. About one third of all the reported cases in these areas were among children at ages 5-9, and more than one fifth were at ages 10-14. Altogether, three fifths of the cases were reported among children under 15 years of age. Very different is the age distribution of deaths from infectious hepatitis. In the country as a whole, in 1951, less than one sixth of the deaths from this disease were at the ages under 15, and more than half were at ages 45 and over. It would appear, then, that the case-fatality rate for the disease is considerably higher for adults than for children.

The recorded incidence rate for infectious hepatitis varies considerably from State to State. In general, the States with the highest case rates are in the northern part of the country. Last year, the greatest number of cases in relation to population was reported in Maine, Oregon, Virginia, and Iowa, where the incidence rate was somewhat over three times the national average of 22 per 100,000 population. In interpreting these results, it should be borne in mind that the extent to which cases are reported is not uniform throughout the country.

National figures indicate that cases of infectious hepatitis occur with greater frequency during the colder months than at other times of the year. From a low point in the summer the number of cases usually rises to a peak in the late winter or spring, and then drops off until it returns to its minimum.

CASES OF INFECTIOUS HEPATITIS‡ THREE STATES§, 1951-1953

AGE (YEARS)	AVERAGE ANNUAL NUMBER	PERCENT OF TOTAL
All ages	705	100
Under 5	42	6
5- 9	225	32
10-14	156	22
15-19	70	10
20-24	43	6
25-29	42	6
30-34	37	5
35-44	41	6
45 and over	49	7

‡Includes serum hepatitis.
§Tennessee for years 1951 through 1953; California, 1951 and 1952; and Oklahoma, 1953.

The control of infectious hepatitis and serum hepatitis presents many difficult problems to physicians and public health workers. While a number of epidemics of infectious hepatitis have been traced to personal contact and to contaminated water and food supplies, the route of transmission in most instances has not been definitely established. There is evidence that the disease may be spread by carriers of the virus who have neither a history of hepatitis nor any clinical symptoms of it. Then, too, knowledge concerning the causative organism is limited because thus far efforts to infect experimental animals with the disease have failed. Furthermore, the disease does not respond to chemotherapy, antibiotics, or any other specific therapy.

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, November, 1954.

†Includes cases of serum hepatitis.

Despite these difficulties, infectious and serum hepatitis are to some extent amenable to control. Gamma globulin has proved effective in preventing cases among persons exposed to the disease. Outbreaks in institutional populations and in camps can be reduced by greater attention to personal hygiene and environmental sanitation.

MORTALITY TRENDS HERE AND ABROAD*

The countries of Western Europe and the English-speaking countries elsewhere experienced substantial reductions in mortality in the period from about 1930 to 1950. The largest declines—approximately one third—were recorded in France and Italy, both of which had relatively high death rates around 1930; the smallest reductions—about one eighth—occurred in Australia and New Zealand, countries with traditionally low mortality. In general, the differences in death rate between countries decreased; this trend is observed for both males and females, and in each age group. Nevertheless, the ranking of the countries according to magnitude of mortality did not change materially in the two decades. The period around 1930 represents the mortality picture just prior to the worldwide economic depression and the political disturbances preceding World War II; the year 1950 is the latest for which a large body of international data is available and is sufficiently long after the close of the war to be generally free from its undue effect upon mortality.

In each of the countries under review, females experienced a relatively greater reduction in death rate than males, even though females generally experienced the lower mortality in 1930. Consequently, the excess of male over female mortality increased during the two decades. In most countries, the excess male mortality in both periods was relatively greatest in the age range 45 to 64 years.

Progress in reducing mortality was consistently more rapid at the younger than at the older ages. At the ages under 45, the death rates in 1950 were generally about half of what they had been two decades earlier. By ages 65 and over, the decreases averaged only about 10 percent; and in a few countries the mortality actually increased somewhat among men at these ages. With this difference in trend by age has come a material change in the age pattern of mortality. The death rate before midlife has been brought down to low levels, while mortality has been concentrated more and more at the older ages.

With respect to level of mortality, the rank of the United States among the countries under survey varied markedly with age. In 1950, very few countries had a better record than ours for mortality in early childhood. The position of our country becomes less and less favorable, however, as age advances. Thus, for white males in the age range 10-34 years, from seven to nine countries showed lower mortality than the United States, reflecting principally the lower incidence of fatal accidents among males at these ages in these countries. By ages 55-59 years, 15 out of the 19 countries had better general mortality records than the United States. The international picture for our white males has not changed essentially over the past 20 years, except that boys at the pre-school ages have improved their relative standing among the countries.

In 1950, white females in our country had a more advantageous position internationally than did our white males at every age period. In only three countries were the mortality rates among white females under age 35 lower than those in the United States. Even at ages 55-59 years, our females occupied a middle position among the countries. During the 20 years under review, the rank of our white females improved at every age, particularly at the extremes of life.

Our favorable mortality experience at the younger age reflects the large measure of control gained over many of the infectious diseases of childhood. Apart

*This article, reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, September, 1954, is based upon a paper, "An International Comparison of Mortality Rates at the Older Ages," by Mortimer Spiegelman, Associate Statistician, Metropolitan Life Insurance Company, presented at the World Population Conference in Rome, August 31, 1954.

12 TREATMENT POSITIONS at your COMMAND...

The Exclusive *Ritter* UNIVERSAL TABLE

In Pediatric work, infant is securely cradled on table.

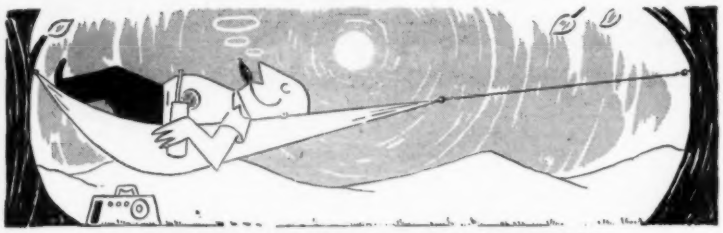
Universal Table in horizontal position for blood pressure check.

"Contour Chair" position with patient comfortably relaxed for nose and throat examination.

...is the answer to all your positioning requirements. Both you and your patients will appreciate the extreme low position of 26½"... eliminates "climbing up"... especially for aged or ailing patients. The 44½" maximum height lets you examine your patients at the "working level" most convenient for you, in any of 12 basic positions. Full 180° table rotation.

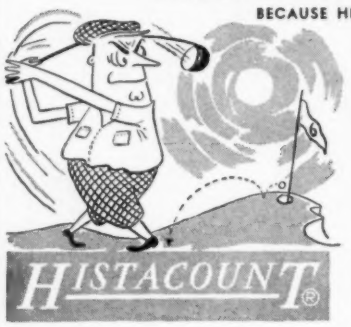
The five-section top has comfortable sponge rubber cushions covered with durable upholstery materials available in 7 standard colors. A light touch of your toe and the motor-driven hydraulic base elevates the table top smoothly, quietly to the position you want. The controls are accessible from either side... saving you valuable time and energy. Then, too, under the Ritter Professional Equipment Plan, you can own this table so easily—for about a dollar per office day. Ask your Ritter dealer for complete details or write the Ritter Company, Inc., Ritter Park, Rochester 3, N. Y.

Ritter Company Inc.



LEISURE, LIVING AND LOAFING

BECAUSE HISTACOUNT KEEPS THE RECORDS STRAIGHT



Hours of desk chores can be easily converted into hammock happiness or a few holes of golf, with Histacount Bookkeeping Systems, Patients' Records and Filing Systems.

Histacount is the symbol of systematic, efficient record keeping which provides the "time off" that Doctors can never seem to find.

Professional Printing Company, Inc.
America's Largest Printers to the Professions.
New Hyde Park, New York.

from our unfavorable accident mortality, it is difficult to explain the relatively poor record of our country at the older ages, particularly for males. It is possible that the relatively low mortality at the younger ages in the United States is due in part to the prolongation of life of persons with physical impairments facilitated by our excellent medical care and high living standards. Consequently, the proportion of impaired lives at the older ages may be greater in the United States than in other countries, with a corresponding unfavorable effect upon mortality. There may also be other and even more important factors at work. It may well be that our poor international standing at the older ages is related to the high proportion of overweight people in our population.

Books Received

PEDIATRIC DIAGNOSIS. By Morris Green, M.D., Assistant Professor of Pediatrics, Yale University School of Medicine; and Julius B. Richmond, M.D., Professor and Chairman of the Department of Pediatrics, State University of New York College of Medicine at Syracuse. Cloth. Pp. 436. Price $10.00. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1954.

HANDBOOK OF TREATMENT. By Harold Thomas Hyman, M.D., author of Integrated Practice of Medicine and Handbook of Differential Diagnosis. Cloth. Pp. 511. Price $8.00. J. B. Lippincott Company, East Washington Square, Philadelphia 5, 1954.

TEXTBOOK OF ORTHOPAEDIC MEDICINE. Vol. I, DIAGNOSIS OF SOFT TISSUE LESIONS. By James Cyriax, M.D., B. Ch.(Cantab.), Physician to the Department

*TWO-FOLD SERVICE—

To The Profession

Professional Foods continue to do their honest best to predicate the needs of the Osteopathic profession in correcting basic and fundamental

NUTRITIONAL TROUBLES

*Since the troubles arise largely in the CHRONIC PATIENT, we have planned our products to aid the doctor of this patient.

*We offer a complete and basic evaluation for the CHRONIC PATIENT at a considerable financial savings in order that treatment for the CHRONIC PATIENT can be directed properly from the start.

Write for added information.

PROFESSIONAL FOODS 219 First St. S.W., Cedar Rapids, Iowa

of Physical Medicine, St. Thomas Hospital, London. Cloth. Pp. 692, with illustrations. Price $12.50. Paul B. Hoeber, 49 East 33rd St., New York 16.

TEXTBOOK OF BIOCHEMISTRY. By Edward Staunton West, Ph.D., Professor of Biochemistry, University of Oregon Medical School; and Wilbert R. Todd, Ph.D., Professor of Biochemistry, University of Oregon Medical School. Ed. 2. Cloth. Pp. 1356, with illustrations. Price $12.00. The Macmillan Company, 60 Fifth Ave., New York, 1955.

HANDBOOK OF EMERGENCY TOXICOLOGY. By Sidney Kaye, B.S., M.Sc., Toxicologist, Office of the Chief Medical Examiner, Commonwealth of Virginia; Associate Professor of Legal-Medicine, Medical College of Virginia; Attending Toxicologist, Veteran's Administration Hospital, Richmond, Virginia; Lt. Colonel United States Army Reserve; Toxicologist, Research and Development Group (Tng.); Lecturer in Legal-Medicine, University of Virginia, Department of Medicine; Formerly Toxicologist, St. Louis Police Department; Toxicologist, United States Army Medical Department; Instructor in Pathology, Washington University School of Medicine; Research Assistant, Toxicology Laboratories of the Chief Medical Examiner, City of New York. Cloth. Pp. 282, with illustrations. Price $5.75. Charles C Thomas, Publisher, Springfield, Ill., 1954.

HERNIA: The Pathologic Anatomy of the More Common Hernias and Their Anatomic Repair. By Chester B. McVay, M.D., Ph.D., Clinical Professor of Surgery and Associate Professor of Anatomy, The University of South Dakota School of Medical Sciences; Surgeon, The Yankton Clinic, Yankton, South Dakota. Cloth. Pp. 40, with illustrations. Charles C Thomas, Publisher, 301-327 East Lawrence Ave., Springfield, Ill., 1954.

STANDARD VALUES IN NUTRITION AND METABOLISM. Being the second fascicle of a Handbook of Biological Data. Edited by Errett C. Albritton, A.B., M.D., Fry Professor of Physiology, The George Washington University. Paper. Pp. 380. Price $6.50. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1954.

APPLICATIONS FOR MEMBERSHIP

CALIFORNIA

Keller, Ethel J., (Renewal) 7207 Alabama Ave., Canoga Park

Haase, Alfred C., (Renewal) 428 W. Fifth St., Chico

Carty, Royal D., (Renewal) 209 Robertson Blvd., Chowchilla

Waldow, Bernard, (Renewal) 2308½ S. Union Ave., Los Angeles 7

MISSOURI

Williams, Lawrence E., (Renewal) 301 Fourth St., Monett

NEW JERSEY

Walker, Elvin E., (Renewal) 222 Berlin Road, Clementon

Ferguson, C. G., (Renewal) 114 Second Ave., Little Falls

NEW YORK

Hitchcock, William L., (Renewal) 21 Mendota Ave., Rye

RHODE ISLAND

Munroe, Robert B., (Renewal) 2247 Mineral Spring Ave., Centredale 11

SOUTH DAKOTA

O'Banion, Harold W., (Renewal) Canton

THE ANSWER IS IN THE ZERO!

YOU can always be sure a TYCOS* Pocket Aneroid is accurate as long as the pointer returns *within zero.* This easy visual check can be made after every reading. And, this Pocket Aneroid can be used in any position. Weighing only 19 ounces, it is ideal for house calls. Fits in a zipper case that can easily be slipped into your pocket or bag. The exclusive hook cuff is designed to fit any adult sized arm and cannot balloon at the edges. It carries this 10-Year Warranty—*manometer readjusted free of charge even if you drop it. Cost of broken parts extra.* See this instrument at your favorite surgical supply dealer. Price **$42.50.** Made by Taylor Instrument Companies. Rochester, N. Y., and Toronto, Canada.

*Reg. U. S. Pat. Off.

Taylor Instruments MEAN ACCURACY FIRST

For HARD, DRY STOOLS OF CONSTIPATED BABIES

Borcherdt

MALT SOUP Extract*

A gentle laxative modifier of milk. One or two tablespoonfuls in the day's formula—or in water for breast fed babies—produce a marked change in the stool.

SAVES DOCTOR'S TIME, TOO!

Fewer phone calls from anxious mothers. Malt Soup Extract is merely added to the formula. Prompt results. Easy for mother to prepare and administer. Does not upset the baby.

*Specially processed non-diastatic malt extract neutralized with potassium carbonate. In 8 oz. and 16 oz. bottles.

BORCHERDT MALT EXTRACT CO.

217 N. Wolcott Ave. • Chicago 12, Ill.

Send for Samples and Literature

GOOD FOR GRANDMA, TOO!

Borcherdt

MALT SOUP Extract*

A New Dietary Management for

CONSTIPATED ELDERLY

A bowel content modifier that softens dry, hard stools by dietary means without side effects.[1] Acts by promoting an abundant fermentative bacteria in the colon, thus producing soft, easily evacuated stools. Retards growth of putrefactive organisms. By maintaining a favorable intestinal flora, Malt Soup Extract provides *corrective therapy for the colon, too!*

DOSE: 2 tablespoonfuls b.i.d. until stools are soft (may take several days), then 1 or 2 Tbs. at bedtime.

*Specially processed malt extract neutralized with potassium carbonate. In 8 oz. and 16 oz. bottles.

1. Cass, L. J. and Frederik, W. S.: Malt Soup Extract as a Bowel Content Modifier in Geriatric Constipation, Journal-Lancet, 73:414 (Oct.) 1953.

Send for Sample

BORCHERDT MALT EXTRACT CO.

217 N. Wolcott Ave. • Chicago 12, Ill.

FOR OLDER PATIENTS...

CHRONIC URINARY INFECTIONS

Urolithia can be given over long periods . . . without toxicity, without irritation, without drug fastness . . . to keep the urine free from E. coli, S. albus, S. aureus. . . . Promptly soothes the irritated membrane while providing bacteriostasis.

DOSE: One tbs. in half cup warm water, q.i.d. ½ hr. a.c. and h.s.

Sample on request

Urolithia® — METHENAMINE URINARY ANTISEPTIC

Cobbe Div., BORCHERDT MALT EXTRACT CO., 217 N. Wolcott Ave., Chicago 12, Ill.

CHANGES OF ADDRESS AND NEW LOCATIONS

Allen, W. Burr, from College Park, Ga., to 16 Stuyvesant Road, Biltmore Forest, Asheville, N. C.

Amalfitano, Louis F., PCO '54; Detroit Osteopathic Hospital, 12523 Third Ave., Detroit 3, Mich.

Anderson, Sibyl Wesson, from Chelsea, Okla., to 314 S. Elson St., Kirksville, Mo.

Arellano, Eugene W., COPS '53; 1218 N. Central Ave., Glendale 2, Calif.

Arvant, William, from Hazel Park, Mich., to 8825 Middlebelt Road, Livonia, Mich.

Axelrod, Alfred E., from Collingswood, N. J., to 49 Haddon Ave., Westmont, N. J.

Babcock, Myron F., from Hollywood, Calif., to 416 W. Sixth St., Los Angeles 14, Calif.

Bailey, Robert V., from Fairview Park, Ohio, to 9246 E. Center St., Bellflower, Calif.

Barnes, Frank Allen, from Glendale, Calif., to 1203 Grandhaven, Alisal, Calif.

Baron, Milton, COPS '53; 3117 Iowa St., South Gate, Calif.

Beech, John E., from First Natl. Bank Bldg., to 409 Sixth St., Perry, Okla.

Bell, A. D., from Denver, Colo., to 540 Carson Ave., Las Animas, Colo.

Bell, M. Lillian, from Atlanta, Ga., to Route 2, Andrea Drive, Lakemont, Ga.

Berkowitz, Sydney, PCO '54; 2561 S. Shields St., Philadelphia 42, Pa.

Bethune, Richard C., from 403 S. Superior St., to 219 Michigan Ave., Albion, Mich.

Braunlich, Donald E., PCO '54; Bay View Hospital, 23200 Lake Road, Bay Village, Ohio

Brockway, David, from Hustisford, Wis., to 116 E. Wisconsin Ave., Pewaukee, Wis.

Brown, William H., from Quitaque, Texas, to Box 311, Idalou, Texas

Buck, George E., Jr., PCO '54; 217 W. Pine St., Grove City, Pa.

Buehler, John L., from 4180 Garthwaite Ave., to 6014 S. Western Ave., Los Angeles 47, Calif.

Burrell, James S., from Los Angeles, Calif., to 724 E. Carson St., Torrance, Calif.

Cash, Phillip S., from Des Moines, Iowa, to 633 21st St., West Des Moines, Iowa

Cavendish, Carl L., from Akron, Ohio, to 613 Fifth Ave., Montgomery, W. Va.

Coan, James E., from 406 Osborn Bldg., to 4159 E. 131st St., Cleveland 5, Ohio

Coan, Richard D., from Tampa, Fla., to 28 S. Spanish St., Cape Girardeau, Mo.

Connell, Ralph M., from 4502 Lovers Lane, to 5101 Ross Ave., Dallas 6, Texas

Conti, Gustave V., from St. Albans, N. Y., to 352 Hempstead Turnpike, Franklin Square, N. Y.

Couch, Richard M., PCO '54; Hospital of Philadelphia College of Osteopathy, 48th & Spruce Sts., Philadelphia 39, Pa.

Crane, Lester L., COPS '53; 2055 Marengo St., Los Angeles 33, Calif.

Crawford, Jack W., from Gainesville, Texas, to 3621 Manana, Dallas 9, Texas

Cyman, John F., from 25545 Ryan Road, to 25525 Ryan Road, Center Line, Mich.

De Lille, George A., COPS '53; Doctors Hospital, Inc., 325 W. Jefferson Blvd., Los Angeles 7, Calif.

Dirlam, K. M., from Massena, Iowa, to Oklawaha, Fla.

Di Stefano, R. G., from 5029 York Blvd., to 4905 York Blvd., Los Angeles 42, Calif.

Doren, Neil C., from Los Angeles, Calif., to Box 97, Morristown, N. Y.

Drapkin, Harold, COPS '53; 4413 City Terrace Drive, Los Angeles 63, Calif.

Drews, Robert O., from 507 Metropolitan Bldg., to 2514 Detroit St., Flint 5, Mich.

Eiceman, George H., KCOS '54; 711 Guilford St., Lebanon, Pa.

Everingham, Donald J., from New Boston, Mich., to 36911 Goddard, Romulus, Mich.

Farnsworth, R. E., from 209 N. Loop Blvd., to 225 N. Loop Blvd., Austin 5, Texas

Fife, Marlin A., COPS '53; 2325 Adams Ave., San Diego 16, Calif.

Fithian, Richard N., from Grove City, Pa., to 105 E. Market St., Mercer, Pa.

Gaston, Samuel W., from Meta, Mo., to 207½ E. First St., Hermann, Mo.

Gautschi, William W., from North Hollywood, Calif., to Community Medical Bldg., 4116 E. Compton Blvd., Compton, Calif.

Gilmore, Jack V., from Cairo, Mo., to Detroit Osteopathic Hospital, 12523 Third Ave., Detroit 3, Mich.

Glaser, Melvin M., from 44 Bell Place, to 980 N. Broadway, Massapequa, L. I., N. Y.

Goldfisher, Joseph S., COPS '53; 722 N. Hayworth Ave., Los Angeles 46, Calif.

Goyette, Robert E., COPS '53; 908 W. Covelle Ave., Inglewood, Calif.

Graham, Frank F., from Winona, Minn., to 1145 Hayes St., San Francisco 17, Calif.

Green, Robert A., from Cape May, N. J., to 8045 N. W. Sixth Ave., Miami 38, Fla.

Greenberg, Gordon, from Trenton, N. J., to 4911 N. Ninth St., Philadelphia 44, Pa.

Gregg, Elizabeth R., from Carnegie, Pa., to 3 Chambersburg St., Gettysburg, Pa.
Hafer, Robert K., from Harrisburg, Pa., to 336 W. High St., Elizabethtown, Pa.
Hamburger, Eugene J., from Olympia, Wash., to 13821 12th Ave., S. W., Seattle 66, Wash.
Hamburger, Rena G., from Olympia, Wash., to 13821 12th Ave., S. W., Seattle 66, Wash.
Harmon, Robert L., Jr., from 1100 N. Mission Road, to 532 Lotus St., Los Angeles 65, Calif.
Harnden, Richard L., from Norwalk, Ohio, to 6590 SOM Center Road, Solon, Ohio
Harrison, Jim T., KCOS '54; Upchurch Bldg., Thomasville, Ga.
Harter, B. P., from Winter Garden, Fla., to Holden Heights Clinic, Orlando, Fla.
Hebard, Kenneth G., from Los Angeles, Calif., to 1623 N. French St., Santa Ana, Calif.
Henry, Walter W., from 2911 W. Ramona Blvd., to 2933 Pyrenees Drive, Alhambra, Calif.
Hocka, Frank J., from Columbus, Ohio, to 8608 Hough Ave., Cleveland 6, Ohio
Hoffman, Lynn V., CCO '54; 741 E. 84th Place, Chicago 19, Ill.
Hollabaugh, Russel E., from Akron, Ohio, to Green Cross General Hospital, 1900 23rd St., Cuyahoga Falls, Ohio
Hutchins, John P., from 420 Naomi Ave., to 6165 Baldwin Ave., Arcadia, Calif.
Imamura, Eugene H., from Seattle, Wash., to 23201 Park Road, Edmonds, Wash.
Ince, William T., from Box 318, to Box 428, Twentynine Palms, Calif.
Irwin, Lowell E., COPS '53; 15 N. Bushnell Ave., Alhambra, Calif.
Jones, Sam P., from 231A Lake Park Shopping Area, to 9104 Garland Road, Dallas 18, Texas
Juday, Lynn R., from Whittier, Calif., to Los Angeles County Osteopathic Hospital, 1100 N. Mission Road, Los Angeles 33, Calif.
Kane, Robert J., PCO '54; Lancaster Osteopathic Hospital, Lancaster, Pa.
Karpman, Benjamin, from 3509 E. First St., to 3500 E. First St., Los Angeles 63, Calif.
Kaupke, John M., from 9631 S. Elmore Ave., to 724 S. Painter Ave., Whittier, Calif.
Kaye, Maurice L., from Indianapolis, Ind., to 326 Locust St., Columbia, Pa.
Kellogg, Theodore M., Jr., PCO '54; 227 S. 17th St., Allentown, Pa.
Kennedy, John, from Glendale, Calif., to 211 W. Las Tunas Drive, San Gabriel, Calif.
Kerwood, Robert I., KC '54; Fort Worth Osteopathic Hospital, Inc., 3705 Camp Bowie Blvd., Fort Worth 7, Texas
Kilpatric, Jay A., KCOS '54; 8638 Trafford Lane, St. Louis 21, Mo.
Kowan, Joel M., COPS '53; 2413 Canyon Oaks Drive, Los Angeles 28, Calif.
Kupper, Victor D., from 3509 E. First St., to 3500 E. First St., Los Angeles 63, Calif.
Lapins, Peter J., CCO '54; 2046 S. 13th Ave., Maywood, Ill.
Larson, Don LeRoy, COPS '53; 2153 N. Tyler Ave., El Monte, Calif.
Lezinski, John L., PCO '54; Grand Rapids Osteopathic Hospital, 1225 Lake Drive, S. E., Grand Rapids 6, Mich.
Little, Earl M., PCO '53; 1817 Windsor Place, Louisville 4, Ky.
Loerke, G. Robert, DMS '54; 1309 Emma Ave., Des Moines 15, Iowa
Loken, Arnold B., from Tucson, Ariz., to 4505 S. Broadway, Englewood, Colo.
Longo, Michael A., from 142 Pine St., to 7 Newbury St., Bangor, Maine
Lowry, William K., from Hollywood, Fla., to 2621 N. Federal Highway, Fort Lauderdale, Fla.
Lyon, Thomas M., from North Sacramento, Calif., to Box 905, Jackson, Calif.
Mahoney, Russell F., KCOS '54; Detroit Osteopathic Hospital, 12523 Third Ave., Detroit 3, Mich.
Marcus, Herbert L., COPS '53; 1415 Lafler Drive, Los Angeles 63, Calif.
Martin, William L., from Aline, Okla., to 129 Southview Drive, Excelsior Springs, Mo.
Matzkin, William L., CCO '54; Portland Osteopathic Hospital, 616 N. W., 18th St., Portland 9, Ore.
Mayer, Richard B., from Des Moines, Iowa, to 187 Maple St., Clyde, Ohio
McBride, Newton E., from St. Johns, Ariz., to McBride Clinic, Ardmore, Okla.
McClary, Robert W., COPS '53; 289 "G" St., Chula Vista, Calif.
Meredith, Ortiz Ray, from 911 Seventh St., E., to 716 Dewey Ave., Nampa, Idaho
Merrill, C. S., from 1501 N. First Ave., to 1501 Highland Oaks Drive, Arcadia, Calif.
Messenger, David L., from 1225 Benito, to 2408 Hagen, Alhambra, Calif.
Miller, Wallace J., from Akron, Ohio, to Green Cross General Hospital, 1900 23rd St., Cuyahoga Falls, Ohio
Milum, Edward W., from Corona del Mar, Calif., to 2641 Circle Drive, Newport Beach, Calif.
Moates, John B., from Benbrook, Texas, to 801 N. College, Tyler, Texas

CAMP
TRADE MARK
supports and appliances

head halter

The new Camp Head Halter is made of strong cream canvas lined with thick fleeced flannel for improved comfort. It is immediately available from your local authorized Camp dealer in both child and adult sizes. Comes complete with spreader bar.

rib belts—men and women

An outstanding feature of the new Camp Rib Belts is their "contour cut" which affords a snug fit and assures uniform support on the full width of the belt, forestalling any tendency to looseness along the bottom edge. Alternating pull straps provide equalized distribution of compression around the complete circumference of the belt. Special locking type buckles are easy to adjust; will not loosen while worn. Shoulder straps, crossed in back, prevent the straps from sliding off the shoulders. Made of 1/8" foam rubber vulcanized on white tackle twill, lined with thick fleeced flannel for greater patient comfort. Immediately available from your local authorized Camp dealer.

S. H. CAMP & COMPANY, JACKSON, MICHIGAN
World's Largest Manufacturers of Anatomical Supports
OFFICES: 200 Madison Ave., New York; Merchandise Mart, Chicago
FACTORIES: Windsor, Ontario; London, England

For APPETITE and GROWTH

Orexin

(STUART)

B_{12} | B_6 | B_1

Softab

For Salt Restricted Diets

CELLU CANNED VEGETABLES

Packed Without Salt, Sugar, or Seasoning

Use Cellu Canned Vegetables to add flavorsome variety to meals . . . Asparagus Tips, Stringless Beans, Peas, Corn, Tomatoes and thirteen other favorites to choose from. Also available, Unsalted Tomato Juice. Food values printed on all labels.

OTHER CELLU SALT FREE FOODS

Canned Rice . . . Soups . . . Canned White Bread . . . Strained Vegetables . . . Mushroom Broth . . . Baking Powder . . . Soyamaise . . . AND MANY OTHERS.

Write for Pamphlet on Cellu Sodium Restricted Foods.

CELLU SPECIAL PURPOSE Dietary Foods

CHICAGO DIETETIC SUPPLY HOUSE Inc.

1725 West Van Buren Street Chicago 12, Illinois

Montgomery, Harold L., COPS '53; Saginaw Osteopathic Hospital, 515 N. Michigan Ave., Saginaw, Mich.

Moriarty, J. J., from 505 Central Life Bldg., to Doctors Moriarty Clinic, 1208 N. LaSalle St., Ottawa, Ill.

Moriarty, Geraldine E., from 505 Central Life Bldg., to Doctors Moriarty Clinic, 1208 N. LaSalle St., Ottawa, Ill.

Moriarty, Lucile M., from 505 Central Life Bldg., to Doctors Moriarty Clinic, 1208 N. LaSalle St., Ottawa, Ill.

Moylan, Patrick J., from Trenton, Mich., to 5565 Allen Road, Allen Park, Mich.

Myers, Henry, from Puente, Calif., to 436 Irwindale Ave., West Covina, Calif.

Nash, Ernest F., CCO '53; 6827 Miller Ave., Detroit 11, Mich.

Norton, Charles W., from 1110 Taraval St., to 680 Sutter St., San Francisco 2, Calif.

Page, Fred J., from 12065 Wyoming St., to 12126 Washburn Ave., Detroit 4, Mich.

Paulson, Joseph W., from 408 Oregon Bldg., to 1611 State St., Salem, Ore.

Pease, G. G., from 31 E. McDowell Road, to 8056 N. 27th Ave., Phoenix 52, Ariz.

Peterson, Walter D., from River Rouge, Mich., to 20010 Ecorse Road, Dearborn, Mich.

Philbrick, Emil A., from 108½ S. College, to 212 S. College, Bethany, Okla.

Pomeroy, Ira L., Jr., from Corpus Christi, Texas, to 1704 Fifth, Bay City, Texas

Rasmus, Charles H., CCO '53; Detroit Osteopathic Hospital, 12523 Third Ave., Detroit 3, Mich.

Ray, Thomas L., from 1805 Fair Bldg., to 554 Summit Ave., Fort Worth 3, Texas

Reves, George Oren, COPS '53; 502 Via Malaga, Newport Beach, Calif.

Riccelli, John A., from Raymondville, Texas, to 108 W. Adams, Falfurrias, Texas

Riegel, Harry I., Jr., COPS '53; 1004 Arroyo Drive, South Pasadena, Calif.

Riedell, Edwin H., from 111 E. Philadelphia St., to 724 S. Painter Ave., Whittier, Calif.

Robinson, Russell E., COPS '53; 2690 Pacific Ave., Long Beach 6, Calif.

Rubin, George S., from Brooklyn, N. Y., to Old Country Road & Floral Ave., Plainview, L. I., N. Y.

Russak, Sidney, PCO '54; Maywood Hospital, 4400 E. Slauson Ave., Maywood, Calif.

Rustin, Garth H., from Paradise, Calif., to 1525 Fulton Ave., Sacramento 21, Calif.

Salkind, Leopold, from 1630 S. Sixth St., to 1530 S. Sixth St., Philadelphia 47, Pa.

Schulman, Samuel Louis, DMS '54; 2200 Longfellow Ave., Detroit 6, Mich.

Seaman, Robert F., COPS '53; 2405 Sepulveda Blvd., Manhattan Beach, Calif.

Secontine, Richard D., CCO '54; Detroit Osteopathic Hospital, 12523 Third Ave., Detroit 3, Mich.

Shelhorse, B. L., from St. Louis, Mo., to General Delivery, Imperial, Pa.

Simmers, M. H., from 106 S. El Molino Ave., to 2118 E. Mountain St., Pasadena 5, Calif.

Spiro, James H., from 1311 E. Lycoming St., to 1100 E. Lycoming St., Philadelphia 24, Pa.

Spivey, Henry A., from 231A Lake Park Shopping Area, to 9104 Garland Road, Dallas 18, Texas

Stager, Frank E., from 142 Pine St., to 7 Newbury St., Bangor, Maine

Stein, Leo, PCO '54; 17 Western Promenade, Cranston 5, R. I.

Steinberg, Harry, COPS '53; 4431 City Terrace Drive, Los Angeles 63, Calif.

Stewart, Harry H., from 3002 Hennepin Ave., to 3548 Second Ave., S., Minneapolis 8, Minn.

Streny, Eugene F., COPS '53; 5322 Via San de Larro, Los Angeles 22, Calif.

Sweigert, Jacob K., from 1829 N. Front St., to 2003 N. Second St., Harrisburg, Pa.

Thaler, David D., from Fairton, N. J., to 630 Sunrise Highway, Babylon, L. I., N. Y.

Thomas, Douglas Coy, KCOS '54; 640B Geoffry, Canterbury Gardens, University City 5, Mo.

Thoreson, John O., from Radio Center Bldg., to New Provident Life Bldg., Bismarck, N. Dak.

Turner, H. W., from 533 Lamont Ave., to 323 Parland Place, San Antonio 9, Texas

Walker, Lee J., from Hillsdale, Mich., to Flint Osteopathic Hospital, Inc., 416 W. Fourth Ave., Flint 4, Mich.

Weaver, Richman G., PCO '54; 8019 Cooke Road, Elkins Park, Pa.

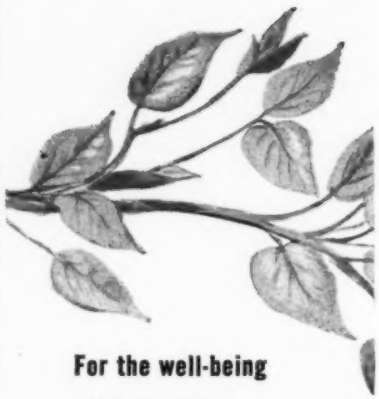
Wiley, Kenneth H., from Atlanta, Ga., to Box 282, Whiting, Iowa

Willard, Margaret, from Lincoln Park, Mich., to 2232 N. Main St., Decatur, Ill.

Wood, Reynold W., COPS '53; 4215 E. Tenth St., Long Beach 4, Calif.

Young, Andrew Roland, KC '54; Stevens Park Osteopathic Hospital, 1141 N. Hampton Road, Dallas 11, Texas

Zwick, William F., from 5138 Third Ave., to 353 N. Mednik Ave., Los Angeles 22, Calif.



For the well-being of your patients

TAMPAX

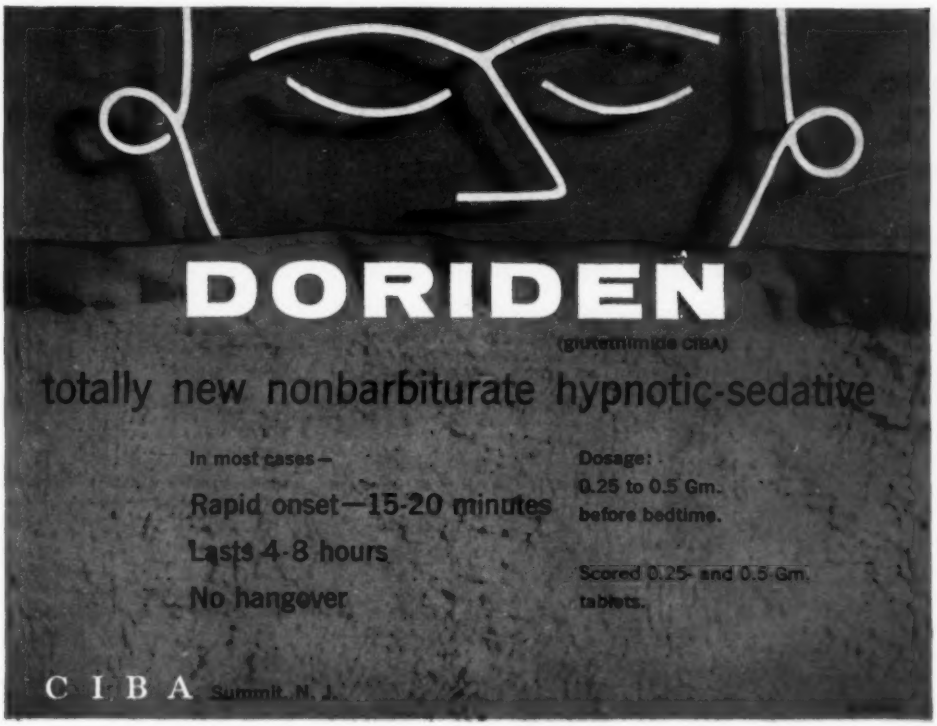
intravaginal protection during menstruation. *Three absorbencies.*

Accepted for Advertising in Publications of the American Medical Association

TAMPAX INCORPORATED

Palmer, Massachusetts

AOA—25



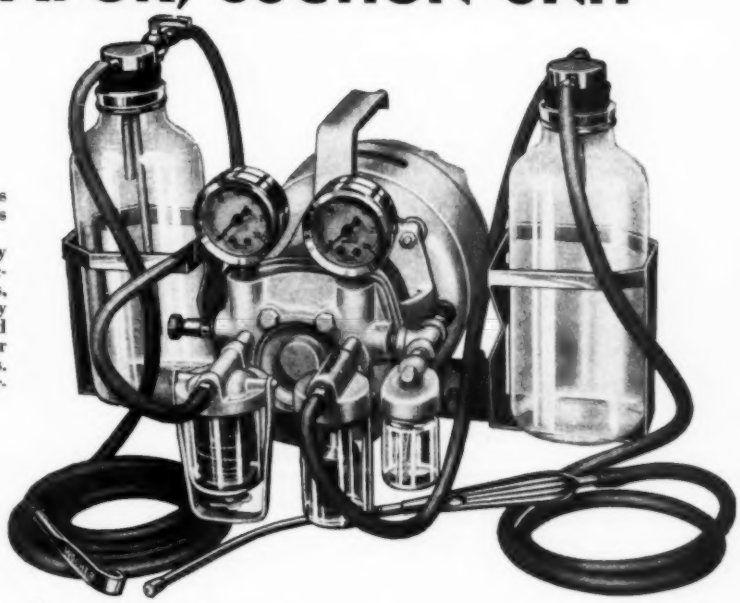
ETHER-VAPOR, SUCTION UNIT

COMPLETE
$99.75

Our "Southwind" Anesthesia Unit comes to you complete—ready to use. There is nothing else to buy.
The ¼ H.P. motor* and 4-blade, rotary pump provides all the pressure and suction you'll ever need. Regulators, Gauges, Muffler, Filter, Automatic Oiler, Safety Trap, Ether Tube, Suction Tube and Rubber Tubing are all included. Motor and pump guaranteed for three years. For 115 V., A.C. 60 cy. only. Other currents slightly higher.

The "Southwind" is an exclusive Wocher product. You won't find a comparable unit anywhere else. The pump only, without suction and ether bottles and tubes, but with regulators, gauges, filter, oiler, etc., is available at $69.50.

*Not explosion-proof.

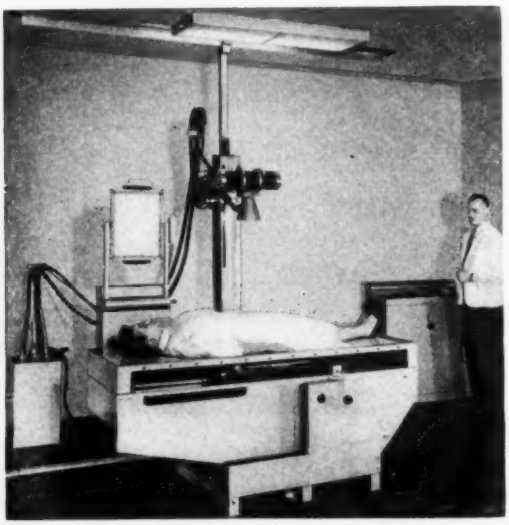


609 COLLEGE STREET **Wocher's** CINCINNATI 2, OHIO

BRANCHES IN DAYTON, COLUMBUS, DENVER, SHREVEPORT

New 200-MA. X-Ray Unit Priced As Low As Comparable 100-MA. Units

Full-Wave Rectified
4 X-ray Valve Tubes in Transformer
100 KVP at All Ma. Settings
Double-Focus Rotating Anode Tube
Fully Automatic Control



Many of the unusual and ingenious features of the control and transformer of this new X-ray Unit were originally in X-ray Generators designed and produced in large quantities by H. G. Fischer & Co. for the Armed Services —all of which were approved by the U. S. Bureau of Standards and performance—proven in service by the Armed Forces. These special features are now available to the Medical Profession in this new, superior, 200-Milliampere X-ray machine at a price as low as many comparable 100-milliampere units.

The unit is available in either 100 or 200 milliampere rating and for single or two-tube operation. A full 100 kilovolts are available at ALL milliampere settings.

The tubestand is furnished in either of two types at the same price—mounted on floor rails or floor-to-ceiling mounted. The tube arm on both types swings laterally through 90° to clear the table for vertical positioning and for single-tube fluoroscopy in both vertical and horizontal positions. A manually operated stereoscopic shift provides a lateral shift of 6" on both sides of center.

The table is precisely counterbalanced for finger-tip tilting. A motor drive of the quiet, but powerful, roller chain type is available. A full-size 12"x16" fluoroscopic screen is mounted on the table. It can be equipped with a spot film device that functions for one central, two horizontal, two vertical, or four corner radiographs on an 8"x10" film.

The control is fully automatic, with its devices aligned progressively from left to right for the setting of each exposure factor in consecutive steps.

The entire unit can be installed in an 8'x11' room with an 8' ceiling height.

Fill in the coupon below for complete information on this new X-ray unit.

H. G. FISCHER & CO.

Franklin Park, Illinois (suburb of Chicago)

Manufacturer of X-Ray, Physical Medicine and Rehabilitation Equipment

H. G. FISCHER & CO., 9451-91 W. Belmont Ave., Franklin Park, Ill.
Please send, without obligation, full information on:.....
☐ FISCHER 200 MA. Radiographic-Fluoroscopic Unit
☐ FISCHER "Spacesaver" Radiographic-Fluoroscopic Unit and Examining Table ☐ 30 MA, ☐ 50 MA, ☐ 75 MA, ☐ 100 MA, ☐ 200 MA.
☐ FISCHER Short Wave Diathermy Units, F.C.C. Approved.
☐ Small Down Payment—Low Monthly Payments—INCOME - AS - YOU - PAY Plan.
☐ FREE Simplified X-Ray Manual.

Name .....AOA
Address..... City..... Zone..... State.....

OFFICIAL AUTOMOBILE EMBLEM

Design, consisting of green cross and gold lettering on white background, is executed in best quality baked enamel on a heavy bronze convex shield. Washable and weatherproof.

Fitted with steel bracket for attachment to license plate holder.

Recognized by many local and state police departments. Supplied only to members of the American Osteopathic Association.

Price $1.50 Postpaid

American Osteopathic Association
212 E. Ohio Street Chicago 11, Illinois

FUNGICIDE
GERMICIDE ANTIPRURITIC

DERMYCIN®

Laboratory and clinical investigations have *proved* Dermycin effective against a variety of skin-infecting bacteria and fungi.
Indications for its use include:

TINEA INFECTIONS
("athlete's foot," tinea capitis, Dhobic itch, etc.)
PRURITUS ANI
(of fungus origin)
ACNE VULGARIS
IMPETIGO
DERMATITIS VENENATA
(as ivy, oak poisoning)
MINOR SURGERY

Dermycin is so useful, so *versatile*, it appeals to specialist and general practitioner alike.

In all cases the area must be washed with mild white soap and water. Dry and apply Dermycin at least twice a day, or as a wet dressing where indicated.

Supplied in 1, 2, 8 and 16 fl. oz. bottles.
(Dermycin is not advertised to the laity.)

Write for professional sample.

CHAL-YON CORPORATION
65 PINE STREET NEW YORK 5, N. Y.

YOUNG'S
RECTAL
VAGINAL
DILATORS

FOR:

- **Constipation**
  Results in 65% of cases without laxatives
- **Anal Stricture, Stenosis**
- **Spasm and Fibrosis**
- **Dysmenorrhea**
- **Post-Operative**
  Hemorrhoidectomy, Rectal Surgery
- **Prolapse**
- **Megacolon**
- **Dyspareunia**

Infants — flexible rubber
Children's and Adults' — bakelite
(Graduated Sizes)

Write for Reprint "Rectal Dilators in Treatment of Constipation"

F. E. YOUNG & CO.
422 E. 75th St., Chicago 19, Ill.

"THE NEAREST APPROACH TO THE CONTINUOUS INTRAGASTRIC DRIP FOR THE AMBULATORY PATIENT".*

NULACIN

A pleasant-tasting tablet...to be dissolved slowly in the mouth...not to be chewed or swallowed...made from milk combined with dextrins and maltose and four balanced non-systemic antacids...**

Promptly stops ulcer pain...holds it in abeyance...and hastens ulcer healing.

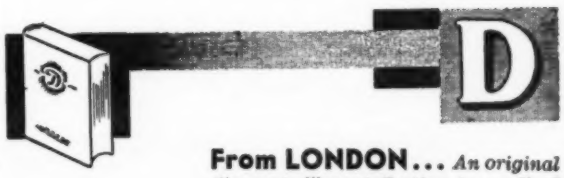
In tubes of 25 at all pharmacies. Physicians are invited to send for reprints and clinical test samples.

Continuous gastric anacidity for prompt relief in peptic ulcer, gastritis, hyperacidity, pregnancy heartburn

*Steigmann, F., and Goldberg, E., J. Lab. & Clin. Med. 42:955 (1953).

**Mg trisilicate, 3.5 gr.; Ca carbonate, 2.0 gr.; Mg oxide, 2.0 gr.; Mg carbonate, 0.5 gr.

HORLICKS CORPORATION
Pharmaceutical Division • RACINE, WISCONSIN



From LONDON... *An original "imported" contribution to medical literature!*

BRITISH OBSTETRIC AND GYNECOLOGIC PRACTICE

NEW! Two distinguished medical editors of London, and 46 eminent British workers in the fields of Obstetrics and Gynecology, present their individual experience in this important contribution to literature. Edited by:

Sir EARDLEY HOLLAND, M.D., F.R.C.S., F.R.C.O.G.
Consulting Obstetric and Gynecologic Surgeon, London Hospital

ALECK BOURNE, M.B., F.R.C.S., F.R.C.O.G.
Consulting Gynecologist, St. Mary's Hospital, London

Here you have a new and completely original work by 46 leaders from every teaching center in England. It truly represents British teaching and techniques as practiced today.

In every section you will see emphasis on the *clinical* and *practical* aspects of obstetrics and gynecology. Pathology is discussed where it bears upon practical diagnostic and therapeutic considerations. Technique is carefully described, with valuable help in 754 illustrations and 13 full-color plates—original drawings, photographs, x-rays, diagrams and colored reproductions. There are several special chapters on the psychology of reproduction, medico-legal aspects, and vital statistics.

The book is COMPLETELY NEW, and is being published simultaneously in England and America by special arrangement. Only a limited supply is available. To be sure you get your copy, order NOW!

TWO VOLUMES, 2022 PAGES 767 ILLUSTRATIONS $29.50

F.A.DAVIS CO.
1914 Cherry Street
Philadelphia 3, Pa.
75 YEARS OF MEDICAL PUBLISHING

F. A. DAVIS CO., 1914 Cherry St., Phila. 3, Pa.

Please send and charge to my account:

British Obstetric and Gynecologic Practice (Two Volumes) . . . $29.50

NAME.....

ADDRESS.....

.....

how many of your patients are eskimos?

Unless you are one of the handful of physicians in the Far North, you'll see an Eskimo only in adventure movies. In a way, it might be a welcome relief to examine a nose hardly ever affected by infectious and/or allergic conditions.

Of course, the estimated 3 out of every 5 patients here in the U. S. suffering from such conditions represent a continuing, and sometimes exasperating, problem.

Now, for these patients, here is a new preparation, effective in 5 out of 6 cases* of

upper respiratory tract involvements
. . . *infectious and allergic*
. . . *seasonal and perennial*
. . . *acute and chronic*

Administration: *Adults*—Two or three sprays in each nostril, 4 or 5 times a day as needed, or as directed by physician.

Children—One or two sprays in each nostril, 4 or 5 times a day as needed, or as directed by physician.

*(Response in 254 of 302 patients[1-5])

1. Busis, S. N., and Friedman, L. L.: Antibiotics & Chemotherapy 3:299, 1953. 2. Lazar, A. M., and Goldin, M.: Eye, Ear, Nose & Throat Monthly 32:512, 1953. 3. Cohen, B. M., and Mendelsohn, R.: Laryngoscope 63:1118, 1953. 4. Wittich, F. W.: Ann. Allergy 12:185, 1954. 5. Vickers, M. A.: Laryngoscope 64:632, 1954.

mucolytic
penetrating

Biomydrin®

antibacterial • antiallergic • decongestant

nasal spray

81107

Nepera Chemical Co., Inc. Pharmaceutical Manufacturers, Nepera Park, Yonkers 2, N. Y.

Advertisers' Index

Abbott Laboratories 23
American Bakers Assn. 16
American Osteopathic Association 60, 68
Ames Company, Inc. 36
Arnar-Stone Laboratories, Inc. 71
Appleton, Century, Crofts 28
Armour Laboratories 4
B. F. Ascher Co. 48

Borcherdt Malt Extract Co. 64
Bristol-Myers Co. ..... Cover II, 12, 26
Burdick Corp. 46

S. H. Camp Co. 65
Chal-Yon Corp. 68
Chatham Pharmaceuticals, Inc. 45
Chicago Dietetic Supply 66
Ciba, Pharmaceutical Products, Inc. ..... Cover IV, 1, 13, 27, 34, 67
Columbus Pharmacal Co. 10

F. A. Davis Co. 69
Desitin Chemical Co. 56

Eaton Laboratories 49

H. F. Fischer Co. 68
Florida Citrus Commission 72

Geigy Pharmaceuticals 31
General Electric Co. 14
Gerber's 33
Otis E. Glidden Co. 23
Gomco Surgical Mfg. Co. 17

Horlicks Corporation 69

Irwin, Neisler & Co. 40

Kinney & Co. 38
Kremers-Urban Co. 39

Lakeside Laboratories, Inc. 32
Lederle Laboratories 7, 58, 59
Lea & Febiger 30
Thos. Leeming & Co., Inc. 42
J. B. Lippincott Co. 22

S. E. Massengill Co. 43
Menley & James 50

Nepera Chemical Co. 70

Ortho Pharmaceuticals 18

Pelton & Crane Co. 44
Pet Milk Co. 24
Chas. Pfizer Co. 9
Professional Foods 62
Professional Printing Co. 62

Ralston Purina Company 20
Riker Laboratories 5, 27, 35
Ritter Company 61
Roerig, J. B. & Co. 11

Sandoz Pharmaceuticals 51
Saunders, W. B., Company ..... Cover I
Schenley Laboratories 25
Schering Corp. 3
Julius Schmid ..... Cover III, 8
Sherman Laboratories 21
Shield Laboratories 37
Smith-Dorsey 41, 47, 53, 57
Martin H. Smith Co. 52
Stuart Co. 15, 66, 71

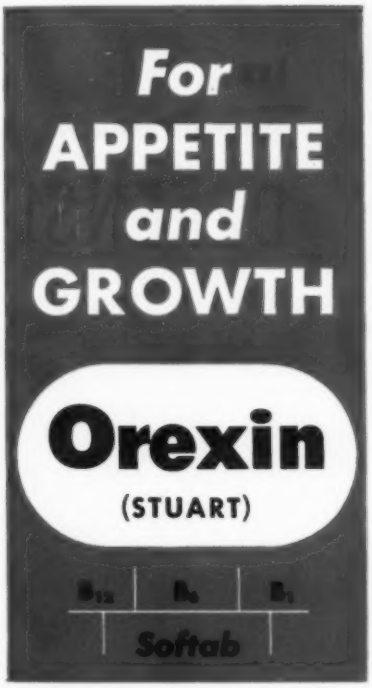
Tampax, Incorporated 66
Taylor Instrument Co. 63
S. J. Tutag Co. 30

U. S. Vitamin Corp. 54

Vitaminerals, Inc. 29

Welch Allyn, Inc. 55
Winthrop-Stearns 6
Wocher, Max & Co. 67
Wyeth Laboratories 19

Young, F. E., & Co., Inc. 69



QUICK RELIEF from Surface Pain and Itching

Americaine AEROSOL

Quick Topical Anesthetic for Office Use

Published clinical reports show nothing relieves surface pain and itching like Americaine . . . because only Americaine contains 20% dissolved benzocaine. Americaine relieves fast, sustains relief from 2 to 6 hours. In Ointment or handy Aerosol Spray for office use. Write for details.

ALSO FOR:
Burns
Abrasions
Hemorrhoids
Post-Episiotomies
Dermatoses

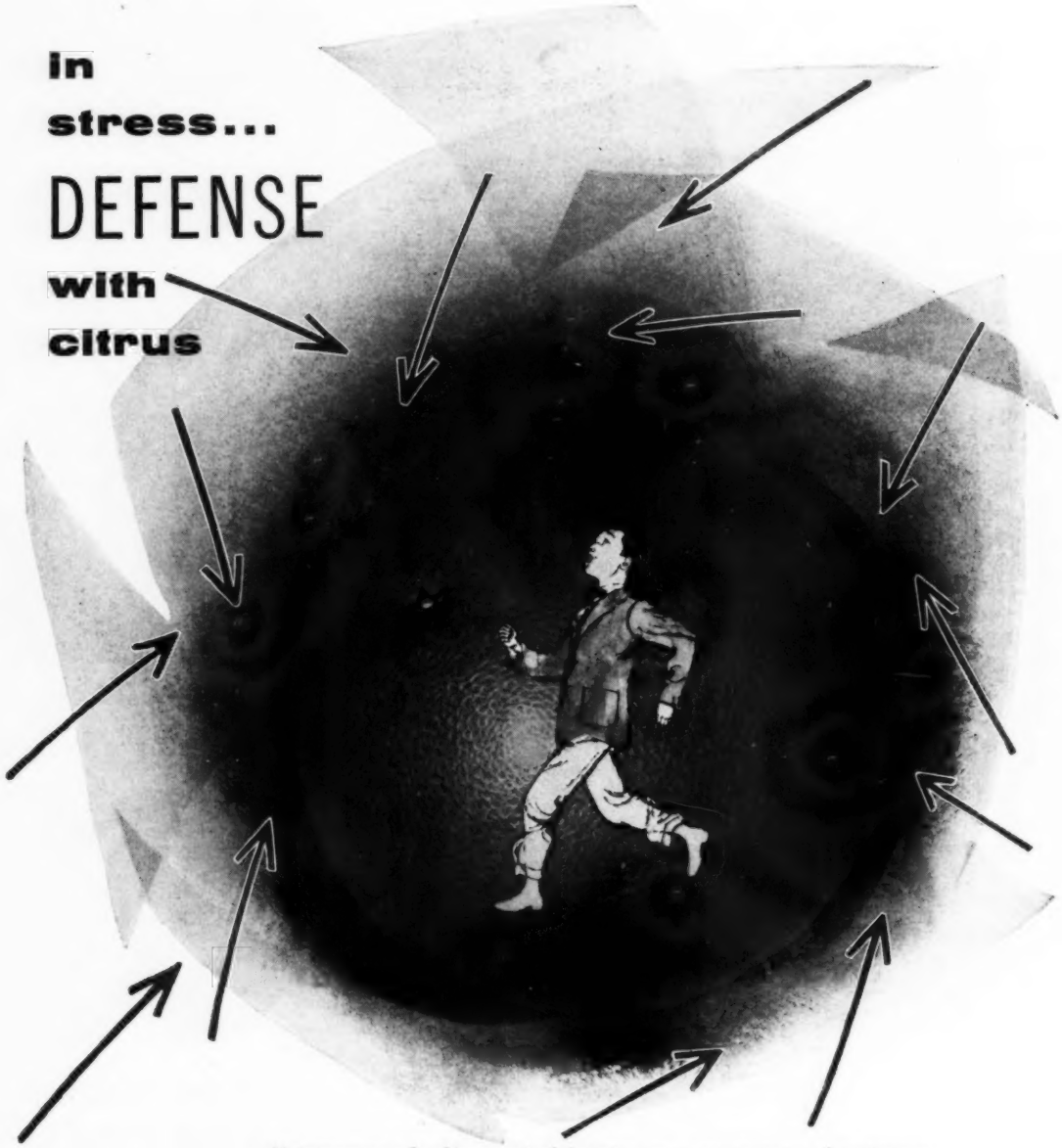
Also Available
Americaine Topical Anesthetic Ointment

ARNAR-STONE LABORATORIES, INC.
1316-C SHERMAN AVE., EVANSTON, ILL.
In Canada: Brent Laboratories, Ltd., Toronto

in stress...

DEFENSE

with citrus



Maintenance of adrenocortical function as a cornerstone of resistance in stressful life situations helps prevent disorders characteristic of the general adaptation syndrome. Since vitamin C is essential to production of anti-stress hormones by the adrenal cortex, a natural defense against stress disorders is an ample intake of readily utilized *natural vitamin* C as provided by citrus fruits and juices.

FLORIDA CITRUS COMMISSION • ***Lakeland, Florida***

FLORIDA *Citrus*

ORANGES • GRAPEFRUIT • TANGERINES

VAGINAL TRICHOMONIASIS

HOW is vaginal trichomoniasis transmitted? Marshalling arguments in support of the sexual route, Trussel points out that "the parasitism is uncommon among children and virgins. Further, the parasite is reported to be most commonly found between the ages of 16 to 35 years, the period of greatest sexual activity."[1] Upton makes a similar observation: "I see very little trichomonas vaginitis in young girls of puberty age, and in older women after menopause."[2]

Travelogue of the trichomonads. If other routes than the sexual are theoretically possible, they are comparatively rare. Trichomonads have been found occasionally in children whose mothers harbored the parasites.[1] Contamination from the rectum is no longer held tenable, since the intestinal and the vaginal trichomonads are "distinct species"[1] and the intestinal species does not survive long in the vagina. Novak says, "Contamination from bath water, or from towels, hands, and instruments must all be considered, though in the individual case the explanation is rarely clear."[3] Trussel states: "Douche nozzles, enema tubes, toilet seats, towels, and physician's gloves and instruments have all been incriminated but never actually proven to spread the protozoa."[1]

Transmitted during coitus. "There is increasing evidence that the organisms are not infrequently transmitted through coitus," says Novak.[3] Trussell lists as additional evidence: the site of the infection in the vagina, the frequent association of trichomonads with gonococci, and the well-known flare-ups during pregnancy and postmenstrually, "when unprotected sexual intercourse is common. . . ." Moreover, Greenblatt and West report that in prostitutes the incidence of vaginal trichomoniasis may be as high as 66 per cent, as compared with 10 to 15 per cent in other women.[4]

Four to nine month regimen for husband. Once the married woman has acquired vaginal trichomoniasis, she may prove "resistant" to treatment, or be "cured," only to relapse. "The vaginal infestation recurs over and over," says Karnaky.[5] In persistent and recurring cases, most

"UNCOMMON AMONG CHILDREN AND VIRGINS"

authorities stress that *the husband is the most likely source of reinfection.*[1,3,5-13] "The real focus has been the male generative organ," continues Karnaky in his recent communication to the J.A.M.A. "In resistant and recurring cases of T. vaginalis infestation the husband should wear a condom at coitus for four to nine months, during which time these trichomonads will usually die out on their own accord."[5] Similar protective measures are recommended by Bernstine and Rakoff[6] and by Trussell.[1]

Take specific measures to win the co-operation of the husband in your treatment of vaginal trichomoniasis. Otherwise he may easily reinfect the patient and nullify the good results of the regimen. In prescribing a condom, be selective and take advantage of Schmid product improvements

When there is anxiety that the condom might dull sensation, the answer is to prescribe XXXX (FOUREX)® membrane skins, made from the cecum of the lamb. These are pre-moistened, tissue-thin and tissue-smooth, and do not retard sensory effect. If cost is a consideration, prescribe RAMSES,® a transparent condom of natural gum rubber, very thin and strong. SHEIK,® also natural gum rubber, is even more reasonable in price.

Your prescription of Schmid brands not only circumvents embarrassment, but assures fine quality. The protection they offer is the very foundation of the re-infection control. Prescribe this protection for as long as *four to nine months* after the wife ceases to show evidence of infestation.

References: 1. Trussell, R. E.: Trichomonas Vaginalis and Trichomoniasis, Springfield, Ill., Charles C. Thomas, 1947. 2. Upton, J. R.: West. J. Surg., Obst. & Gynec. *60*:222 (May) 1952. 3. Novak, Emil: Textbook of Gynecology, ed. 3, Baltimore, The Williams and Wilkins Company, 1948. 4. Greenblatt, R. B., and West, R. M.: Antibiotics in the Treatment of Vaginal Trichomoniasis, paper presented at the 2nd Annual Antibiotic Symposium, Washington, D. C., October, 1954. 5. Karnaky, K. J.: J.A.M.A. *155*:876 (June 26) 1954. 6. Bernstine, J. B., and Rakoff, A. E.: Vaginal Infections, Infestations, and Discharges, New York, The Blakiston Company, 1953. 7. Crossen, R. J.: Diseases of Women, ed. 10, St. Louis, The C. V. Mosby Co., 1953. 8. Kanter, A. E.: Postgrad. Med. *12*:457 (Nov.) 1952. 9. Karnaky, K. J.: Urol. & Cutan. Rev. *42*:912 (Nov.) 1938. 10. Lanceley, F., and McEntegart, M. G.: Lancet *1*:668 (Apr. 14) 1953. 11. Reich, W. J., et al.: J. Internat. Coll. Surg. *16*:86 (July) 1951. 12. Reich, W. J., and Nechtow, M. J.: Practical Gynecology, Philadelphia, J. B. Lippincott Co., 1950. 13. Rogers, G. C., and Fleming, J. M.: Am. J. Obst. & Gynec. *68*:563 (Aug.) 1954.

JULIUS SCHMID, INC., *Prophylactics Division*
423 West 55th Street, New York 19, N. Y.

Nasal

congestion

relieved

as long as

6 hours

Effective and physiologically compatible nasal vasoconstrictor.

Solution 0.05% in 1-fluidounce bottles with droppers, and in pints.

Solution 0.1% in 4-fluidounce bottles.

Jelly 0.05% in 20-Gm. applicator-tip tubes.

Nebulizer, 15 ml. Privine hydrochloride Solution 0.05%.

Also available: Privine Ophthalmic Solution, 0.1%, in 15-ml. bottles with droppers.

Privine® hydrochloride (naphazoline hydrochloride CIBA)

CIBA
Summit, N. J.

2/20864